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LISTER'S LAST ASSISTANT

by

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WHEN LORD LISTER died it was universally acknowledged that the King had lost his most distinguished subject and the world its greatest inhabitant. Nobody is more conscious of the truth of this statement than myself. This knowledge makes the attempt to describe and estimate his character and attainments for the benefit of future generations—a task beyond my biographical powers. The greatness of this man must not be measured by what appears to me, now that it is written, only a superficial impression of an almost unearthly superhuman being.

Yet for all that, I was probably in closer professional contact with him than anyone during the last four years of his active professional life. Sir Watson Cheyne became too busy in his private practice to continue as Lister's assistant, and as I was then Cheyne's assistant in his laboratory and private practice, it seemed quite natural that I should undertake his duties and become Lister's assistant. This I did, and assisted him in many operations both in his hospital and private practice. I also had charge of his cases when he went away.

During my association with him we had many conversations that were not entirely concerned with professional matters. I once asked him if it were true that when at the bedside of a case of simple fracture of the femur he had said, " Gentlemen, you may have noticed that simple fractures such as this unite without any complication, but when the skin is broken, as in a compound fracture, then the course of the case is quite different, and fraught with the utmost danger. Erysipelas, pyæmia, and hospital gangrene often threaten the safety of the limb, and even life of the patient. The man who discovers why this happens and can prevent it, will make his name and fame in surgery." He answered that the story was quite true.

Apart from all his other scientific investigations, this problem dominated his thoughts and actions. The train of thought was laid and only needed Pasteur's work to act as the match in order to ignite it. The result was that he was able to proclaim to the world that to secure primary healing of a wound it was necessary to prevent the entrance of germs. This

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discovery immediately revolutionized surgery, introduced bacteriology into medicine, and made experimental and fundamental operations on animals possible. The necessity of preventing germs entering or infecting wounds is as urgent to-day, as it was when first announced.

The introduction of antiseptic surgery was made easier by the fact that in ordinary septic wounds, Lister was dealing with non-spore bearing micro-organisms. To demonstrate this fact the late W. Bulloch F.R.S performed the following experiment. He soaked strands of silk in cultures of the spore bearing Hay Bacillus. He then immersed them in a solution of 1-20 Carbolic Acid, and perchloride of Mercury in various strengths. The tubes containing them were hermetically sealed, and kept intact for twenty-seven years. At the end of this time, after neutralizing the chemicals, he placed the strands of silk in culture media. Growth of the Hay Bacillus occurred in every culture.

On one occasion Lord Lister told me that Queen Victoria had written to him and asked him, as President of the Royal Society, to do all in his power, by speaking and writing, to stop vivisection. He knew that his reply would be very disappointing to the Queen. He sent a very carefully worded reply, saying that it was a vital necessity for the good of Her Majesty's subjects that vivisection should be allowed to continue. He further respectfully assured the Queen that vivisection in this country was under the careful supervision and control of Her Majesty's Government. After receiving this letter the Queen took no further notice of him.

On another occasion he bitterly complained, saying, " When going to the hospital I sit in my carriage, thoroughly depressed, as I know I shall lecture to about ten students, whereas I used to give the same clinical lecture to hundreds in Glasgow and Edinburgh." During my tenure of office as surgical registrar, I attended every clinical lecture he gave and then realized what an inestimable advantage it would have been to the teaching of surgery in London if he had been the professor in charge of the London University surgical clinic, instead of being merely one of a dozen or more surgeons, teaching surgery in various hospitals each of which was trying to be a separate University Clinic.

He used to come into the lecture theatre, sit down, and with his semi-closed fists resting on his thighs, deliver his lectures to his meagre audience. There was none of the dogmatic certainty displayed by other lecturers in surgery, who were only imparting information which could be read in the text books. Everything he said was illustrated by thoughtful experiment from his own original conceptions and experiences which included

and elucidated many of the problems that concerned surgery. Inflammation, coagulation of the blood, the details involved in the preparation and sterilization of catgut ligatures and other suture material; the advantages and possible explanation of the benefits of counter irritation, were all subjects upon which he lectured. In his lecture on anæsthesia he was particular to point out the safety of chloroform administration provided the respiration and not the pulse of the patient was the principal consideration. He also demonstrated that the normal behaviour of pigment cells in the frog depended upon the reflex action of the nervous system, the afferent impulses of which were conveyed by the optic nerves, and, that in areas of inflammation the nervous mechanism was temporarily paralysed.

After telling us of his successful experiments in proving this he would add, " Curiously enough I was the first to point this out."

I took careful notes of all his lectures but I will not publish them because they would not equal in interest and information all that is contained in the two volumes of " The Collected Papers of Joseph Baron Lister." These contain the gist of all his clinical lectures, and should be read by all candidates preparing for their final examinations. They are informative and make the reader think and realize how a great intellect works.

For so tall and manly a figure, his voice was a comparatively small one, although when occasion demanded, he could be heard by the most distant of his listeners. No man attending his lectures or operating theatre could fail to be imbued with the same reverence for his profession, as so palpably influenced every word or deed of Lister himself. The tone of his lecture room and operating theatre created the same reverence as should be experienced on entering a church. Everyone there worked silently and quietly. No one spoke except Lister himself and everything he said was for the good and safety of the patient. He knew everything that was occuring in his theatre.

On one occasion he had asked me to supervise the administration of chloroform by one of his clerks. While he was in another part of the theatre the loud respirations of the patient suddenly stopped. I knew all was well, but Lister rushed to the operating table and only when he found everything was satisfactory, did he allow me to continue.

On another occasion I was assisting him to wire a fractured patella, and for a moment he thought I had handed him the wrong end of the wire; his comment was, "If it had been wrong I would never have forgiven you."

On yet another occasion I pushed a blade of a Spencer Wells forceps through a very thin intercostal wall into the pleural cavity. He immediately plugged the opening with a periosteal flap which he turned down from the rib. I expected a reprimand, but nothing was said.

Another and last example of the sanctity with which he regarded his work is as follows. He had begun an operation for supra-pubic lithotomy but found the relation of the peritoneum such that the bladder could not be opened without injuring this membrane. He at once determined to perform a lateral lithotomy and extracted two calculi in thirty-two seconds with such dexterity that the onlookers could not refrain from cheering. Lister indignantly turned round and said, " Gentlemen, gentlemen, remember where you are."

About the time of which I am writing, the surgeons of London had accepted the truth of Lister's teaching, but failed to carry it out with all his precautions. They had abolished pyæmia and hospital gangrene from their wards, but suppuration was far too common and it was not fully realized by them as being the disastrous result of inefficient technique. There was no text-book on bacteriology—in fact only Lister, Cheyne and a few others knew anything about this subject—and it was only when surgeons became more educated that operation wounds healed more frequently by first intention.

The sort of thing that happened was that after sterilizing his hands, the surgeon would handle the notes and temperature chart, and read to his audience the nature of the case upon which he was going to operate, and then proceed without re-sterilizing his hands. At another time a surgeon would ask for an instrument which had not been sterilized and the " instrument maker " would take it from the cupboard, and hand it to the operator for use without being sterilized.

In fact at one meeting of a medical society a well-known surgeon was anxious to show Lister a case upon which he had performed excision of the knee joint. Unfortunately for the reputation of the operator, Lister was shown a badly suppurating wound. He turned to me saying, "Merely a septic case," and passed on. I only hope that it was a lesson to the surgeon in question. It was this kind of transgression that made me regret that Lister had abolished the spray. Its continuation, unnecessary for Lister, would have been an additional precaution to all those whose antiseptic technique was imperfect.

He was the most accomplished and dextrous surgeon I have ever met, and always seemed to be guided by an uncanny instinct. He instinctively

saw at once what should be done and proceeded to do it with an unerring judgment that was always right. I never saw a case of his " go septic." Yet according to our modern ideas we should regard his methods as inefficient. He operated in his everyday clothes, keeping his hands moistened with 1-20 Carbolic solution. Beads of perspiration appeared at once on his forehead and chin immediately he commenced to operate, but not when he dressed his cases.

From his benevolent and gracious manner it was surprising that he fought for his principles, but he did so, in his own way. First, by example, and secondly, by his public addresses to professional bodies. In these he often laid stress on the usual lapses in Antiseptic technique.

For example, in an address on " The present position of Antiseptic Surgery " which he delivered in Berlin in 1890 before the International Medical Congress, he included the following allusion to Bantock and Lawson Tait who claimed success without the use of Antiseptics. He said, " In truth the practice of these surgeons is by no means conducted without Antiseptic precautions."

The only criticism he made was their habit of washing out the peritoneal cavity with ordinary tap water, although Bantock had had the water boiled before use. It is not true to say that these men introduced what is now called " Aseptic Surgery." In Lister's clinical lectures he often stated that boiling water was the best antiseptic, and he used to tell us he had conducted surgical operations without the use of Antiseptics. But when he found that only about half a dozen men knew anything about bacteriology, he introduced Antiseptics to assist those surgeons who were ignorant of the careful precautions necessary to keep germs out of wounds.

There were then no text books on bacteriology in this country. Except on Lister's first operation day, no London surgeon attended to learn his technique, but on the first day the theatre was full to see him wire a fractured patella. I heard one of these first-class surgeons say, " It was a foolhardy and risky performance." On the next operation day and for ever afterwards only his house surgeon, dressers and a few students were present.

The disappointment to Lister was immense. Occasionally a French or a German surgeon came round the wards with him, and they had the case explained to them, each in his own language.

Lister's tall, striking figure and broad shoulders dominated any occasion, by his presence. His correspondingly large head was completely covered

by iron-grey hair which was inclined to curl. His whiskers were white. His forehead was broad and well formed. His handsome features were small and regular. He very rarely smiled. He once was very much amused by a man whose head bandage was kept well in place by a safety pin which had accidently pierced his ear. Upon being asked why he had not complained he answered, " I thought you did it on purpose Sir Joseph."

He attended the hospital daily and on Sunday mornings he walked to the hospital from the Temple Church where he was a regular attendant. Apart from his epoch-making scientific discoveries he was, as I have said, the most dextrous surgeon I have ever seen, as well as being an accurate, careful and successful diagnostician. He was accused of being a slow operator. He purposely did not hurry because he said the chief cause of shock was the sepsis which too often followed operations performed by others. If he wished to cover a raw surface by a skin flap, then it was perfectly planned and accurately fitted to the new surface.

A test of extreme accuracy and care is the operation for the cure of a complete Cleft Palate and Hare Lip. In these operations he was always successful. When dressing a wound he would often hand the dressing which had been next to the wound to an onlooker, so that he could satisfy himself that there was no smell from it.

His epoch-making discoveries speak for themselves and for all time. In these few remarks I have endeavoured to show the reverence with which he regarded his work and with which he inspired all who came into contact with him. As a teacher and operator he has had no equal.

I can only conclude by saying how proud I am to know that Lord Lister trusted me, and that I assisted him at the last operation he performed.

W. E. HENLEY and LISTER

W. E. HENLEY, THE POET and Essayist, was crippled from boyhood as the result of tuberculous osteitis and had one foot amputated. In 1873, when amputation was recommended for his remaining foot, he decided to seek the help of Joseph Lister—"a young surgeon then beginning to be famous." He travelled to Leith by sea from Wapping and then by train to Edinburgh, and was admitted to the Royal Infirmary where he remained under Lister's care for 20 months. In letters to Harry N—— (a keeper of a coffee-house), he wrote of Lister : " He is a great surgeon, my boy! Antiseptic surgery his theory and practice, will have to fight its way, to fight for life." . . ."Joseph Lister is an Englishman and (whether he save my foot or no) we may rejoice therefor. The conceit of these bloody Scotchmen is something atrocious."

Henley describes how Lister was followed on his rounds by a crowd of students which "looked like the ring, seen from behind, round a conjurer."

Henley's more serious pen-portrait of Lister has often been quoted and is well-known. It was first published with the title of "The Surgeon" in

the Cornhill Magazine in July, 1875, among a series of verses with the heading "Hospital Outlines—Sketches and Portraits." These were published later in his book of Verses and Henley's appreciation of Lister then appeared under the title of " The Chief " in the series "In Hospital: Rhymes and Rhythms."

" THE CHIEF."

His brow spreads large and placid, and his eye
Is deep and bright, with steady looks that still.
Soft lines of tranquil thought his face fulfil—
His face at once benign and proud and shy.
If envy scout, if ignorance deny,
His faultless patience, his unyielding will,
Beautiful gentleness and splendid skill,
Innumerable gratitudes reply.
His wise, rare smile is sweet with certainties,
And seems in all his patients to compel
Such love and faith as failure cannot quell.
We hold him for another Herakles,
Battling with custom, prejudice, disease,
As once the son of Zeus with Death and Hell.

HORMONES

Lecture delivered at The Royal College of Surgeons of England

on

15th July, 1947

by

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To ATTEMPT TO COVER the field of hormones in one hour is of course an impossible task and one can only just give a general picture of the present position, emphasizing the more modern developments.

The most important organ in the endocrine system is the anterior lobe of the pituitary gland. Its importance was recognised as soon as the operation of hypophysectomy on laboratory animals became an ordinary standard practice. The immediate effects of hypophysectomy differ according to whether the animal has passed the period of puberty. The hypophysectomised pre-puberal animal ceases to grow and sexual development does not occur. The hypophysectomised mature animal shows characteristic changes in the reproductive system. In the female complete cessation of the œstrus cycle occurs, whilst in the male atrophy of the testes and external genitals occurs immediately together with loss of potency and spermatogenesis.

By a long and elaborate series of researches the connection between the anterior lobe of the pituitary and the other endocrine glands has now been worked out and the following list of hormones indicates very briefly the present state of knowledge.

1. *Growth Hormone.* This is responsible for the growth of the animal up to its full mature stature. It is also interesting to note that whilst by and large the hypophysectomised immature animal does not grow, this is not strictly speaking accurate, since a certain small increase in weight takes place. This is known as somatic growth and is due to the inherent power of .the individual parts of the body to increase in size. Administration of an extract containing the growth hormone would cause a rapid increase in the rate of growth of the hypophysectomised animal and it will reach its normal maximum without difficulty. The administration of the growth hormone to immature intact animals will accelerate their rate of growth until the maximum is reached. Giant animals have never been produced by the administration of growth hormone. This is believed to be due to the production of an anti-hormone. Whilst various clinical reports have been made showing that such preparations have caused growth in dwarf children, there is no really potent preparation of the growth hormone on the market at the present time.

2. *Lactogenic Hormone.* This fraction of the anterior lobe of the pituitary is responsible for stimulating the mammary glands to secrete

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milk. The gland must first of all have been prepared by the action of the œstrogenic hormone. This of course occurs in pregnancy. The common test object of the lactogenic hormone is the growth of the crop glands in the pigeon. The use of the lactogenic hormone clinically has been described and favourable results have been reported of its use in conditions where milk secretion in the human subject is deficient.

3. *Gonadotrophic Hormones.* It is now generally accepted that two gonadotrophic hormones are produced in both male and female pituitary glands. These are known as follicle stimulating hormones and luteinising or interstitial cell stimulating hormones. The follicle-stimulating hormone is present in the serum of pregnant animals and is prepared commercially from pregnant mares' serum. The luteinising hormone is excreted in the urine of pregnancy and is often known as chorionic gonadotrophin.

In the female the function of the follicle-stimulating hormone is to produce growth in the follicles of the ovary up to the pre-ovulatory stage. In the male this hormone causes the growth of the semniferous tubules.

The luteinising hormone in females produces ovulation and the formation of the corpus luteum and also stimulates the interstitial cells of the ovary. In the male, in addition to stimulating the interstitial cells, it stimulates the Leydig cells to produce androgens.

Commercial preparations of both hormones are available and have been used with some success in the treatment of amenorrhœa, functional uterine bleeding and sterility. The preparations are extremely potent and must be used with great caution, frequent examinations being made to guard against untoward effects. In the male their use is rather limited and results have been disappointing. Luteinising hormone has been used in the treatment of undescended testes and follicle-stimulating hormone in the treatment of various types of male infantilism such as Frohlich's syndrome.

The anterior lobe of the pituitary also secretes a series of other trophic hormones affecting the thyroid, parathyroid, the suprarenal cortex and the pancreas. These hormones are of great theoretical interest but have not reached the stage of clinical application. They. are tested for by observation of rehabilitation of the particular gland in question in the hypophysectomised animal. The gland showing the greatest change, apart from the gonads, in hypophysectomy, is the cortex of the suprarenal. This diminishes in size very rapidly and in certain hypophysectomised birds death from cortical insufficiency can occur.

We now turn to the hormones produced by the individual glands. It is not necessary to consider the hormones of the suprarenal medulla, thyroid, posterior pituitary, &c., since nothing new has been added to our knowledge of these substances in recent years. The most striking advance has been in what is now known as the steriod hormone field. We can consider these hormones separately under the headings of the ovary, the testes and the suprarenal.

Throughout the 1920's attempts were made by many workers to isolate the active substance from the ovary. Allen and Doisy in 1923, showed that the extracts could be tested by the vaginal smear technique of Stockard and Papanicolaou on ovariectomised rodents. A great impetus was given to the work in 1927, when Aschheim and Zondek showed that the urine of pregnant animals contained large quantities of the ovarian hormone. By 1930, three groups of workers had isolated in crystalline form substances which would produce œstrus in ovariectomised female rodents. The general formula was shown to be as follows :—
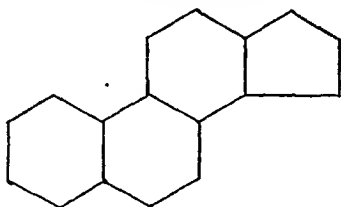
The two most common œstrogenic substances occurring in nature are œstrone and œstradiol which have the following formulæ :—
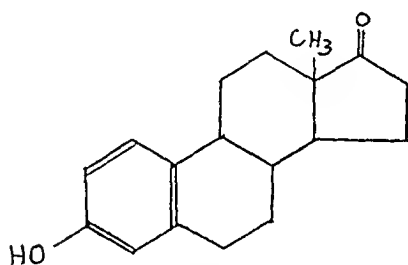
OESTRONE
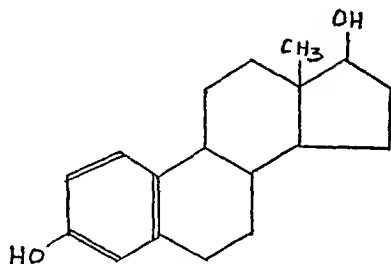
OESTRADIOL

These substances are generally administered in the esterified form in order to prolong their action. They are almost inactive when given by mouth and are, therefore, always given by injection.

Owing to the extremely complex nature of these substances their synthesis in the laboratory is at present out of the question. However, the fact that more than one substance can be found in nature having the same qualitative action gave rise to the idea that it might be possible to find synthetic analogues of simpler structure which would have œstrogenic activity. A long series of experiments was undertaken, preparing and testing compounds first of the phenanthrene type with the following general formula :—

2
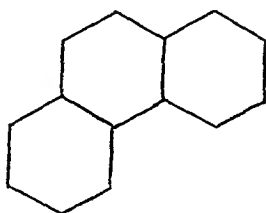
and later of even simpler substances having only two rings. Finally, the three compounds, stilbœstrol, hexœstrol and dienœstrol were made. Their formulæ are as follows :—

STILBOESTROL

HEXOESTROL

DIENOESTROL
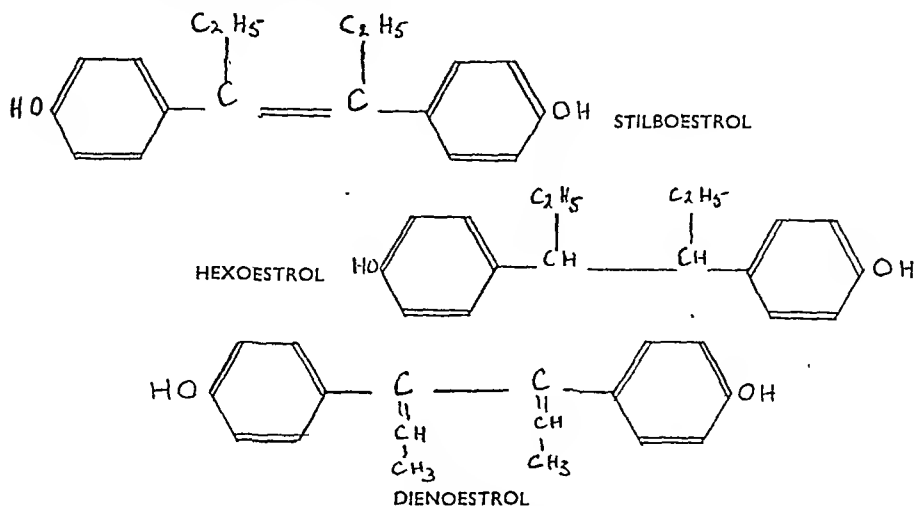
These were found to be capable of replacing the naturally-occurring œstrogens in every way, and also to be active when given by mouth. For this reason they are generally used in clinical practice in preference to the naturally-occurring œstrogens.

The chief clinical use of œstrogens is in the treatment of the menopause. Very small doses, often as low as 1mg. a day, will suffice to control the symptoms. Œstrogens are also given for the suppression of lactation where this is necessary and for various gynæcological disorders where these are thought to be due to a deficiency of œstrogens. Certain side-effects, such as nausea have been reported from the use of the synthetic œstrogens. These are not serious as a rule and often pass off even if the dose is maintained. It is probable that the side-effects are bound up with the œstrogenic action and are analagous to the nausea which occurs in the first few months of pregnancy when the system is flooded with œstrogens.

The most dramatic use of œstrogens has come to light in recent years. This is in the treatment of carcinoma of the prostate. Their use in this condition is due to the work of Huggins who, in 1940, tried the effect of massive doses of œstrogen as an alternative to castration in a number of advanced cases. This treatment has now become a standard practice and it can be said that the large majority of cases show some response. One of the earliest symptoms of this form of cancer is frequency due to urinary obstruction. This is followed in due course by intractable pain due to pressure on nerves. These symptoms are often relieved in a striking manner by the administration of œstrogens, so that patients who have been bedridden and in acute pain and discomfort have been able to get up and resume their normal activities. The response is also shown by a return to normal levels of the acid serum phosphatase and in many cases

the primary tumour and secondaries are found to have decreased in size. A certain proportion of cases which respond at first do escape from control later and it is not possible to say at present why this is. As to the mode of action of œstrogens in carcinoma of the prostate, it is most likely that the action is on the anterior lobe of the pituitary, which is known to be inhibited by the action of œstrogens, and that by this means the action of androgens, which act on certain cells of the prostate gland, is also inhibited.

Œstrogens have also been used for the treatment of carcinoma of the breast and a small number of these cases have responded favourably.

It is not possible to produce menstruation by the administration of œstrogens alone. This does produce uterine hæmorrhage, but curettage of the endometrium shows that the characteristic changes have not taken place. It was suspected for a long time that the ovary produced another hormone in addition to the œstrogenic, and this was proved by Corner and Allen, who in 1929, demonstrated the presence of a hormone in the corpus luteum. This hormone, progesterone, has the following formula :—
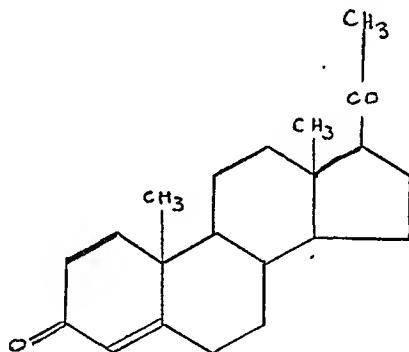
PROGESTERONE

It is used in the treatment of menorrhagia and also in the treatment of habitual abortion and some success has been reported.

The same difficulty of isolation was found with the male sex hormone until it was shown that large quantities of androgenic substances are excreted in the urine of male animals. The first of these substances, androsterone, was isolated from urine by Butenandt. This has the following formula :—

ANDROSTERONE

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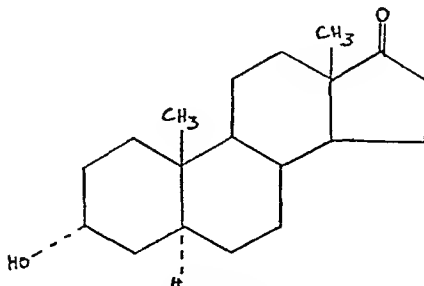
Later a much more active substance, testosterone, with the following formula :—
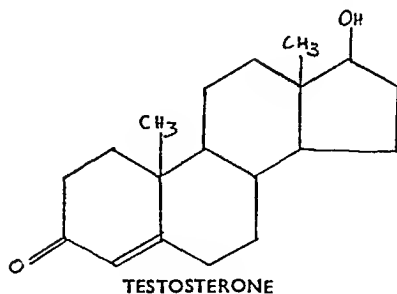
TESTOSTERONE

was isolated from bulls' testes. Testosterone has an extensive clinical application in the treatment of acute testicular deficiency following castration by either trauma or surgery. Male characteristics and potency can be completely restored by the administration of testosterone subcutaneously. It is, however, of no value in impotency which is not associated with signs of hormonal deficiency, the modern view being that such impotency is of psychogenic origin. The continental literature before the war contained extensive references to the use of testosterone in the treatment of " male climaterium." This, however, is a condition which appears to be confined to the Continent.

The clinical use of both progesterone and testosterone has only been made possible on its present extensive scale by the work of Ruzicka who showed that these substances can be obtained from cholesterol by a process of chromic oxidation which removes the long side-chain at carbon atom 17-, and subsequently by a shifting of double bonds, &c. This enables the active substances to be isolated from raw materials easily procured in large quantities and a large industry for the manufacture and sale of various steroid hormones has now been built up. Soya beans are used as the starting material.

Another hormone belonging to the steroid group is that of the adrenal cortex, known as corticosterone, with the following formula :—
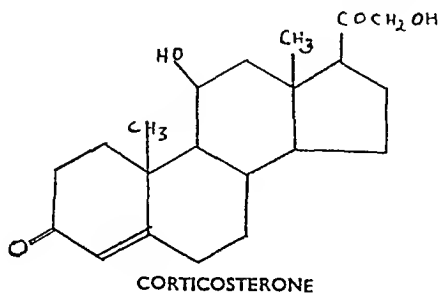
CORTICOSTERONE

A more active compound, and one which can be obtained by the same process of sterol degradation as that used for progesterone and testosterone, is desoxycorticosterone. This is used in the treatment of Addison's

disease with complete success provided the right dosage is maintained. Its use in the treatment of shock has been disappointing.

The assay of the various hormones excreted in the urine has been found a useful aid to diagnosis. The Aschheim-Zondek and Friedman tests for pregnancy are of course well known and universally employed, but there are also some pathological conditions where a hormonal assay of the urine is of great diagnostic value. For instance, it has been found that in patients, especially females, with adrenal cortical tumours the excretion of androgens is excessively high. By measuring the amount of androgen excreted in the urine it is possible to distinguish between cases of adrenal tumour and Cushing's syndrome, as in the latter case the excretion of androgens is either normal or sub-normal. Again, it is possible to distinguish between various types of testicular tumours by differentiating between the types of gonadotrophic hormone excreted in the urine. Thus the seminomatous type of tumour is associated with an increased output of follicle-stimulating hormone, and this type is radio-sensitive, whereas cases of mixed epitheliomata are often associated with an output of chorionic hormone, and these cases are resistant to X-rays. The hormonal assay, therefore, is not only a guide to diagnosis, but also enables the doctor to decide on treatment and to forecast its possible success.

REFERENCES

It is not considered necessary in a discursive paper of this type to give detailed references to all the original papers, but full details and references will be found in the following two books :—

CAMERON (1947) *Recent advances in endocrinology.* 6th edition. Churchill, London.

BEAUMONT and DODDS (1943) *Recent advances in medicine.* 11th edition. Churchill, London.

THE PATHOLOGY OF SUDDEN DEATH

Lecture delivered at the Royal College of Surgeons of England

on

29th July, 1947

by

Keith Simpson, M.D.

Lecturer in Forensic Medicine to Guy's Hospital

THIS REVIEW OF the more common pathology of sudden death is based on an analysis of my own medico-legal case records of 15,000 autopsies. These range from simple natural deaths to violent wilful killing, and cover all ages from birth to that of a man who claimed 108 years—a slight exaggeration into which he had been flattered by Press attentions.

The proportion of deaths from natural causes was 68 per cent.—roughly two-thirds—a reminder of the vast amount of ordinary morbid anatomy passing through the hands of the forensic pathologist. Among these natural deaths there figured some 41 per cent. of the total 15,000 —a little over 6,200 cases—where death took place suddenly, if not instantaneously, upon collapse, an unexpected event precipitating death within a few hours of apparent good health. It is this group which I have extracted for study, and I make no apology for concentrating on the principal common disorders rather than the rarer—if more diverting—diseases.

I have excluded the hazards of delivery at birth, such as atelectasis of the lung, and developmental defects which could not properly be included within the orbit of disease, for these are problems in other fields.

Some prefatory remarks should be made with regard to the wording of the cause of death, for here a personal factor must enter. Where, for instance, senility, hypertension and coronary occlusion by atheroma exist together at death—as is common enough—it is reasonable to assume that all three have contributed to death. The heading used for such cases has depended upon the relative contribution assessed for each disease, and in such a matter I have to ask you to trust to my evaluation —right or wrong. As age increases, its contribution to the decompensation of a heart which has held hypertension at bay for many years must gradually increase also : it must become increasingly reasonable to say that senility is the cause of such a death. However, this is a human whim,

not measurable, and which makes broader statistics of this kind unsuitable for mathematical niceties.

The field covered is indicated by the table :—

ANALYSIS OF MORE COMMON NATURAL CAUSES OF SUDDEN DEATH

Cause of Death		Pathological Analysis
Abortion (natural)	10	(Immediate death only)
Acute Adrenal insufficiency ..	15	
Allergic shock	4	
Aneurysm (aortic) rupture ..	143	Syphilis 80 per cent.
		Atheroma 20 per cent.
Aorta rupture (other than aneurysm)	160	Hyaline 60 per cent.
		Atheroma 30 per cent.
		Syphilis 10 per cent.
Asphyxia (natural causes) ..	140	(cf. unnatural causes 93 per cent.)
	26	
Cerebral hæmorrhage ..	806	Cerebral 75 per cent.
		Pontine 14 per cent.
		Cerebellar 11 per cent.
Subarachnoid hæmorrhage ..	268	Developmental aneurysms 78 per cent.
Cerebro-spinal fever	50	
Coronary embolism	10	
hypoplasia	11	
occlusion	638	Ostial occlusion (sy.) 7 per cent.
rupture	7	
thrombosis	988	
Heart infarction	517	
rupture	264	
hypertensive failure ..	484	
Heart valvular disease ..	472	Rheumatic 79 per cent.
		Syphilitic 21 per cent.
		Aort. sten. 36 per cent.
		Aort. insuf. 28 per cent.
		Mitr. sten. 20 per cent.
		Mitr. insuf. 4 per cent.
		Compound 12 per cent.
Hæmoptysis (var. causes) ..	36	
Hæmatemesis (var. causes) ..	73	
Influenzal pneumonia	222	
Pneumothorax	9	
Pulmonary embolism	317	Recent injury 25 per cent.
Senility	577	
	6,247	

Fig. 1.

It is a striking feature of the complete table that, of the common natural causes for sudden death, the failings of the coronary or cerebral arteries alone account for over 56 per cent. These, unlike the failings of the aorta or of the valves of the heart, rest upon the ravages of disease or of what Clifford Allbutt has called the " qualities of tissues carried away by the

stealthy hours " for which medicine has no known treatment—a sobering thought which might well stimulate new interest in the pathogenesis of arterial sclerosis.

Now let me draw attention successively to some of the more interesting data.

Acute Adrenal Insufficiency

In 1937, in an Erasmus Wilson lecture, I discussed the pathology of this fatal syndrome, finding examples of the diseases recognized to erase the functioning adrenal with enough rapidity to cause death within a few hours. The most common is undoubtedly bilateral spontaneous hæmorrhage, and, whereas those which occur after infancy are still recognized as being largely due to meningo-coccal or streptococcal septicæmia, the former in cerebro-spinal fever as the Waterhouse-Friderichsen syndrome, those which occur in the few days or weeks after birth have subsequently been recognized to be commonly due to erythroblastosis fœtalis—and not to the exaggerated hyperæmia of the androgenic zone in the post-natal period. Acute caseous tuberculosis and malignant growth deposit comprise the majority of the remainder. Willis (1933) drew attention to the frequency of mass bilateral deposits, especially from the bronchus and the breast, and many observers have confirmed his figures of some 9-10 per cent. of all cancerous spread including the adrenal gland—and as commonly both as one.

Rupture of the Aorta (Without Aneurysm)

This condition is as common without aneurysm as with it, indeed increasingly more common, and the stimulus given by T. Shennan in his original monograph on the subject (M.R.C. Special Report No. 193, 1934) has resulted in a more critical examination of many ruptures which might at first sight appear to be due to the atheroma that is so likely to be present (whatever the cause of rupture) after the age of 40. Hyaline change—fatty atrophy of the medial muscle, mucinoid and hyaline degeneration of the connective tissue and fragmentation of the elastin—is more common in men (65 per cent. males : 35 per cent. females in Shennan's original series) and, like all vascular ruptures, aggravated by any degree of hypertension. The ascending aorta gives way more commonly than any other part, some four out of five of my own 160 cases bursting between the aortic valve and the origin of the innominate artery. Hyaline degeneration was responsible for 60 per cent. of these. The reportage of dissecting aneurysm has lagged to a remarkable degree behind that of rupture without aneurysm, for the sole reason that although some degree of dissection is almost invariable—usually between the media and adventitia—it is usually short, being cut short by rupture into the pericardium. Nevertheless, some remarkably long dissections have been recorded, and I have on many occasions traced the course of a rupture which has commenced in the aorta into the upper thighs.

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" Twin-bore " aorta malformation, with two intimal lined channels, is an interesting phenomenon which illustrates the possibility of healing, and a case of healing of the primary split which occurs in the present series has already been reported in the English literature—*Lancet* 1940.

Sub-Arachnoid Hæmorrhage

By far the most common natural cause of this catastrophe is rupture of a developmental aneurysm situated on the circle of Willis, or, infrequently, on one of its critical branches. In a recent paper on the subject, Magee, Principal Medical Officer to the Ministry of Pensions (1943), refers to Symonds' original pointer (1924) on the frequency of the condition *when seriously looked for*, and reports the finding of 43 aneurysms in 58 autopsies for sub-arachnoid hæmorrhage, solitary in all but five cases, and on the front half of the circle of Willis in these times as frequently as the back half. In my own series for this lecture 78 per cent.—rather more prove to have such an origin. Nothing short of stereoscopic dissecting glasses will provide adequate sight for some of the minute aneurysms. When no dilation can be found, perfusion is the only sure method of locating the leak. The use of the word " congenital " is unfortunate for such aneurysms, for they are not present at birth; it is the microscopic anatomy of the cerebral vessels whose development is responsible and " developmental " is preferable. Glynn (1940) and Carmichael (1946) have both drawn attention to the important defects of elastin in the internal lamina in the cerebral vessels or distinct from those in the media which occur almost as commonly in many other parts of the body. Of the other factors which contribute to rupture, we cannot discount—a rise in blood pressure, either in the form of hypertension or from exertion. A number of cases in the present series have occurred during moments of maintained tension as, for instance, in a young garage hand who lay under a car tightening a nut with a spanner, fit one second, crying " Oh ! my head," the next, collapsing and unconscious, the spanner clattering to the floor immediately after. He was dead when a doctor arrived some four or five minutes later. A word of caution on the interpretation of these aneurysms later in life. There may be deposits of fat and lipoids in their walls, and, of course, some degree of atheroma may, with increasing years, be developing in the circle of Willis. It must not be lightly assumed that saccular aneurysms in such company are of a different pathology : they are likely to be identical in origin. Atheroma of the cerebral vessels results in a different form of dilatation, not saccular, but bulbous, fusiform and occurring along the course of affected vessels, not from the stems of their branches.

Coronary Artery Disease

In coronary occlusion lies a cause—perhaps the only natural cause apart from senility—for literally *instantaneous* death. Coronary occlusion

by ostial specific disease, atheroma of the course of the vessel or throm-
bosis in addition, rarely rupture, accounted for no less than 26 per cent.
of the total of sudden deaths and infarction, or rupture, depending on
such disease, for a further 12 per cent. of this series. It is remarkable that
this disease should only have come into proper recognition as late as
1910 or 1912, mainly through Herrick and Mackenzie, for it undoubtedly
existed in the time of the famous clinico-pathological observers of the
1820-60 period—Addison, Wilks, Bright, Gull and Hodgkin, to mention
only one group at a famous London Hospital. In the Gordon Museum
of that hospital there were displayed specimens of " myomalacia cordis "
—due to coronary artery disease. They were plainly softened infarcts.
Rupture was also shown—an event which, except for trauma, I have not
seen in nearly 20,000 autopsies unless through an infarct or infarct scar.

The sex frequency and distribution of coronary thrombosis in a series
of my own cases was reported to the Medical Society of London in 1939.
I shall concern myself here with only one aspect of the disease—its
immediate pathogenesis. Numerous observers, notably Winternitz
(1938) and Nelson (1942) have drawn attention to the frequency of subin-
timal hæmorrhage as a factor in the genesis of thrombosis : the key to the
lesion is the vascularisation of the normally avascular intima. When
atheroma develops, the new sinusoidal capillaries which appear are most
liable to rupture. Rupture is likely during violent exercise or upon
direct trauma, and the diffusion of thromboplastic substances is a likely
consequence of subintimal hæmorrhage, or the moreso of one which
ruptures the diseased intima. Any interruption of the smooth flow of
blood by obstruction of the lumen is naturally likely to foster the develop-
ment of thrombi—especially during periods of rest or collapse from any
cause.

As regards trauma causing the development of thrombosis, this con-
ception of the pathogenesis is of the greatest importance. Warburg's
valuable monograph (1938) on the non-penetrating injuries of the chest
extracted 197 cases among which are a number associating trauma and
coronary thrombosis in a manner which would satisfy searching criticism
though naturally not providing exact scientific proof. Stern's remarkable
" Trauma in Internal Diseases " adds to these a number of similar cases
whose pathogenesis has been equally critically tested.

Asphyxia in· Infants

Here is a problem to be handled with the greatest care : it is indeed
largely a matter of technique. There is no doubt that bronchitis or
bronchiolitis—a common cause of infant death—can develop in as little
as 12 or 18 hours (Hubble and Osborn), the child appearing normal when
fed, placed in a cot or bed and dying overnight from the accumulation
in sleep of muco-pus in the air passages. Thelin of Zurich claimed (1942)
that in 20 out of 30 cases he examined because the suddenness of death
had precluded certification—even raised suspicion—he could demonstrate

blocking of the lumen by inflammatory secretions. Unfortunately his criteria were not sufficiently critical. During the process of any asphyxiation the lungs become congested and excess mucus, œdema fluid and even blood will accumulate in the air passages. Only microscopy can detect whether pus also lies in this material. Worse, vomiting may complicate the issue, *resulting from* asphyxia and adding yet another fluid to that ahead, in the air passages. It is at this point that a serious error commonly arises : the death is attributed to the inhalation of vomit—when, in fact, the vomit is no more than a reflection of a state of asphyxia already present. Of Thelin's series of 30 cases, seven (*i.e.,* 23 per cent.) died of suffocation. It is likely that, applying the criteria of microscopy to the fluid in the air passages the proportion of cases would be higher. In the 233 cases of asphyxia in infants extracted for this survey, no fewer than 93 (40 per cent.) were due to accidental suffocation.

The application of more strict criteria virtually doubles the number, excluding as non-inflammatory many air passage fluids that might incautiously be described from their naked-eye view as inflammatory. Thelin also gave a flicker of life to one now virtually dead bogy status lymphaticus when he attributed death to this " state " in three of his 30 cases. The status lymphaticus is no more, in fact, than a status, and if we admit, however unwillingly, the possibility (Campbell, 1937) that such subjects may die the more readily of some trivial cause *it does not excuse the failure to find that cause.* We should be no more at liberty to say that a person died of flat feet or a dwarf stature—or some other status.

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ANOXIA IN ANÆSTHESIA

Post-Graduate Lecture delivered at The Royal College of Surgeons of England

on

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by

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THIS IS A tangled and difficult subject in which issues are not clear cut and very much depends upon the careful definition of the degree and duration of the oxygen shortage under discussion.

It is rather easy to erect a hypothesis whereby death is always explained as finally due to anoxia of some tissue or organ, and all disease to lesser degree of the same disorder. Therefore, it is not surprising that "anoxia" is frequently advanced as the explanation of unsatisfactory anæsthetic situations. Very often it may be a valid explanation but at other times it confuses rather than explains.

It is again rather easy for the physiologist to say that on no account may oxygen shortage be permitted during anæsthesia and that the only thing which needs to be said upon the subject is "prevent it." Unfortunately, the clinical anæsthetist knows this command to be impossible of fulfilment. A patient may have organic disease of the lungs and circulation ; he is, therefore, anoxic when conscious. The surgical operation perhaps, requires that he be placed in a position which hinders the respiratory movements almost as though it were designed for this purpose, and when the pleural cavity is opened the situation deteriorates further ; the patient is now necessarily more anoxic. Finally the anæsthetic agent, even under ideal administration, may depress the respiratory response and promote secretions which occlude the air passages. Useless to say "avoid anoxia"— the clinical anæsthetist must try to determine the degree of anoxia present and assess the duration of it which can be permitted without serious outcome, immediately, or as a result of delayed effects of this bad experience amongst all the factors which determine the course of the patient's convalescence.

Again, the anæsthetist may administer a gaseous anæsthetic agent so weak that he can only secure a sufficient partial pressure of it in the lungs by diminishing the pressure of oxygen below the normal figure. "Don't do such a thing," might be the physiologist's advice, but the clinical anæsthetist knows that he can only avoid this "physiological insult" by inflicting the "pharmacological insult" of using a more toxic agent—and the clinical anæsthetist must make the difficult choice.

One possible method of discussing the subject is to examine first the effect of oxygen shortage upon the major physiological systems of the

intact organism and to study the effect of this anoxia in normal, unnarcotised subjects. The way in which anoxia arises during anæsthesia may then be examined, and from this the method of combating the deficiency will emerge. Finally, there will remain a great many open questions upon which we have at present far too little knowledge.

The Effect of Anoxia on Respiration

Acute fulminating anoxia, such as is seen when pure nitrogen is suddenly breathed, causes initial hyperventilation followed rapidly by irregular gasping breathing and very soon respiratory arrest. This effect is of little interest to anæsthetists.

Acute anoxia—such as occurs when an unnarcotised individual is exposed to an altitude of say 30,000ft. (or breathes at sea level a mixture containing 3 per cent. to 4 per cent. of oxygen), causes marked hyperventilation. The increase may be great—as much as 30-60 litres/min. It is not dependent upon carbon-dioxide retention, and indeed the alveolar carbon-dioxide tension falls markedly and the apparent or alveolar respiratory quotient becomes very great.

These changes may perhaps be regarded as the very first step in "acclimatisation" and they tend to reduce the degree of the anoxia. Normally, the pressure of oxygen in the alveolar air is considerably below the pressure in the inspired air, but owing to the increased "mixing" during hyperventilation the alveolar oxygen pressure rises toward the inspired oxygen pressure. Again the decrease in alveolar CO_2 pressure allows the alveolar oxygen pressure to rise a little more, a factor which is quite significant when the anoxia is due to low total barometric pressure rather than low oxygen percentage.

The mechanism of this hyperventilation is not fully understood, but it is probable that it is not due to a direct effect of the anoxia on the nervous breath-regulating mechanism in the central nervous system but to reflexes arising from the chemo-receptors in the carotid sinus and perhaps the aortic arch. The central mechanism—the so-called respiratory centre—appears to be responsive only to changes in blood pH or CO_2 tension. In deep anoxia or narcosis, or a combination of both, it becomes unresponsive so that respiration is then predominantly, so to speak, "anoxia-driven." If in these circumstances the anoxia is abruptly relieved, respiration temporarily ceases.

Lesser degrees of anoxia than that mentioned do not produce well-marked changes in respiration but at any pressure of oxygen in the inspired air below 80-90 mm. Hg. some increase in respiration becomes noticeable and the alveolar carbon-dioxide pressure slowly falls. This effect has been noticed clinically by those anæsthetists who permit some rebreathing, and thus increase the pressure of carbon-dioxide in the inspired air, during nitrous oxide anæsthesia.

Not much more can be said about respiration unless we carefully define the conditions of observation. During nitrous oxide anæsthesia the respiration has been carefully and elegantly studied.

If the following conditions are present,

(a) Nitrous-oxide/oxygen are being given alone.

(b) There is no obstruction to respiration.

(c) There is no surgical stimulus *or* the patient is known to be too deeply anæsthetised to show reflex response to it, then

increasing deficiency in oxygen supply is shown progressively by :—

(1) Increasing rate of respiration.

(2) Prolongation and "forcing" of the expiratory phase.

(3) Irregularity of rhythm.

(4) Slowing of respiration.

(5) Cessation—reversible by immediate inflation of the lungs with oxygen.

Circulation

The reactions of the circulation to increasing anoxia depend again upon whether the degree and rate of onset are great or moderate, and upon whether the subject is recumbent or vertical.

Anoxia of moderately severe degree and rapidity of onset in a recumbent subject results in an initial increase in cardiac output—a compensatory action rather like the hyperventilation already discussed. This increase may be $2\frac{1}{2}$-3 times, and may place a serious strain upon a diseased heart. The coronary blood flow increases as does the blood pressure, pulse volume and pulse rate. The increased blood pressure is maintained until or shortly after the cessation of respiration.

Arterioles in the skin constrict but prolonged anoxia leads to capillary dilation in other organs, loss of fluid and protein through the capillary walls and inter-cellular œdema. The effective circulatory blood volume decreases.

Anoxia of very gradual onset is not usually accompanied by a rise in blood pressure—on the contrary the blood pressure falls gradually from the onset until the final sudden drop as respiration ceases.

Similarly in the vertical human subject or in a subject who has diminished blood volume the full-bounding pulse and high blood pressure are liable to be absent even during rather severe anoxia—instead a slow fall in blood pressure is seen. There will be occasion to refer to this response again later.

Central Nervous System

The first group of signs and symptoms which anoxia produces in the function of the C.N.S. are all reversible. They are, inattention, lack of concentration (rather than lack of the ability to concentrate, because in the early stages, at all events, sufficiently powerful motivation can overcome the effect of anoxia), lack of self-judgment and ability to assess performance, euphoria, or irritability, loss of muscular co-ordination and

lack of muscular power, absence of insight into one's own condition, jactitations, increasing drowsiness and finally unconsciousness.

If before, at, or immediately after the loss of consciousness an oxygen supply is restored, the signs and symptoms are quickly abolished. If, however, the subject is allowed to remain unconscious for some time without restoration of the oxygen supply he may suffer irreversible changes which fall into one of the following clinical groups.

1. Convulsions, respiratory failure and immediate death.

2. Prolonged coma, after restoration of oxygen supply, usually with vomiting, headache, intermittent convulsions, incontinence, paresis, effusions in serous cavities, hyperpyrexia and death hours or days later.

3. Symptoms as in group 2, but with partial recovery. There may remain personality changes which are difficult to assess except by those who know the subject well, or more objective mental changes of all grades of severity up to that of an incontinent partially paralysed individual, apparently without sensory perception.

Such severe sequelæ generally follow either periods of a few minutes during which effective circulation has ceased, or more prolonged periods of grave anoxia while circulation continued. Cases in group 3 have occurred in patients who suffered a severe circulatory reaction to the injection of local anæsthetic and whose "collapsed" condition continued for two hours before restorative measures were effective. Cases in group 2 have followed inhalation anæsthesia in which the anoxia did not arouse the anæsthetist's attention, even though there is good reason to believe it was present.

Cases in all three groups have occurred during emergencies in flight and in the course of experiments in aviation medicine. In these circumstances one can be morally certain that anoxia alone was the prime cause of the condition.

During the war considerable experience was accumulated of the reactions of fit, normal young men to anoxia, and it may be of value to endeavour to summarise some of this experience in terms perhaps of value to anæsthetists for comparison with their clinical observations.

Variations in Response

1. Normal young men can survive considerable exposure to altitudes of 15,000ft. (428 mm. Hg.—equivalent to approximately 11·8 per cent. oxygen in inspired air at 760 mm. barometric pressure) without loss of consciousness. Vision in dim light is the first function to be affected and after about one hour, muscular inco-ordination may appear.

If such normal young men are exposed to a barometric pressure corresponding to 25,000ft. (282 mm. Hg. equivalent to approximately 8 per cent. oxygen at 760 mm. barometric pressure) unconsciousness supervenes in a few minutes. The speed of onset of unconsciousness is increased, of

course, if the subject takes exercise. Immediately after the onset of unconsciousness the subject's pulse is found to be normal in rate, slightly increased in volume, and his systolic blood pressure normal or slightly elevated. If oxygen is now administered recovery is prompt although if pure oxygen is given the so-called "paradoxical effect" of oxygenation may be seen, in which convulsive movements, nausea, vomiting or syncope occur *after* the administration of oxygen and as the subject's blood oxygen-content is apparently increasing.

2. A second group, variously estimated as from 2-10 per cent. were classified as " fainters." These men, subjected to increasing anoxia do not show a gradual deterioration of mental and muscular functions, but appear surprisingly unaffected until quite suddenly they suffer a syncopal attack with rapid thin pulse, low blood pressure, pallor, &c. From this they may recover, even though their oxygen supply is not increased. It is difficult to be sure that other factors (for example, emotion, increasing volume of intestinal gases, &c.) are not also operative, yet in many cases it would seem likely that anoxia alone is responsible. Any impairment of cerebral circulation renders this form of response more probable.

3. Another small group of subjects was encountered who seemed at the time of exposure to be very resistant to anoxia. Many showed marked hyperventilation. Judged upon clinical signs alone they could not be said to be deeply anoxic, although from instrumental measurements one knew they were breathing a very low pressure of oxygen.

After the exposure these subjects sometimes showed serious sequelæ— prolonged headaches, vomiting, visual impairment, muscular weakness and chest pain being some of the symptoms which persisted up to 48 hours.

Thus the worker in this branch of aviation medicine, perhaps in contrast with the anæsthetist, anticipated a certain number of cases in which sudden syncope would occur during anoxia, and also recognised a group of cases in which the immediate physical signs provided a poor guide to the degree of anoxia or of its effect upon the body if sequelæ are taken into account.

Limits

It is known that men have survived at 20,000ft. (equivalent to approximately 9 per cent. oxygen at 760 mm. Hg. press.) for several hours, though death has been reported in these conditions in less than one hour.

At 32,000ft. (equivalent to 5 per cent. oxygen at 760 mm. Hg.) death in less than 15 mins. has occurred.

At 30,000ft. (equivalent to approx. 5·5 per cent. oxygen at 760 mm. Hg.) anoxia is sufficient to bring on unconsciousness in about 2-3 minutes and most aviation physiologists would, I think, consider it hazardous to leave a subject unconscious for more than three minutes in these conditions before restoring a full oxygen supply.

The importance of the shape of the oxygen dissociation curve for blood was fully appreciated in aviation medicine.
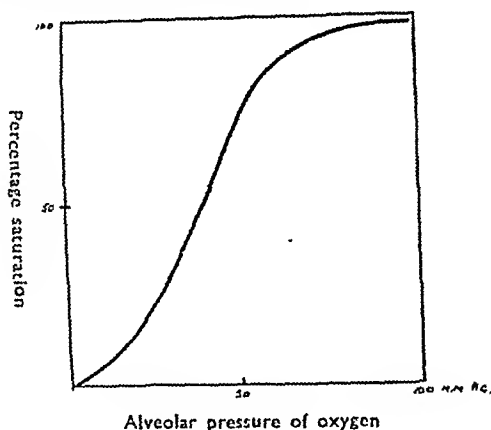
Fig. 1.

Fig. 1 shows an approximate oxygen dissociation curve for human blood. It will readily be seen that an alteration of the alveolar pressure of oxygen in the region of 75-100 mm. produces little change in the degree of saturation of the blood, and thus of the oxygen available to the tissues.

A similar alteration of the oxygen pressure in the region of 50-60 mm. will produce a much greater alteration however, because the slope of the curves is much more nearly vertical in this region. It is thus possible, without causing serious confusion, to refer to the percentage of oxygen in inspired air in a rather approximate manner, in the region of 12 per cent. or above. To refer to "four or five" per cent. however, is a statement which conveys almost no information as, owing to the critical effects of small changes in this region, much greater precision must be observed. It may be that some of the difficulty which exists in reconciling the experience of anæsthetists with that of other workers in this field arises from the difficulty of observing the composition of inspired mixtures with the necessary precision using existing methods of administration.

The Forms of Anoxia in Anæsthesia

The four classical forms of anoxia described by Haldane all occur during anæsthesia and to these one subdivision may be added.

A. *Anoxic Anoxia*

This is by far the most frequently occurring form of anoxia during anæsthesia and therefore the most important.

The primary defect is that the alveolar air contains too small a pressure of oxygen and as a result the proportion of the hæmoglobin in the blood which leaves the lungs in an oxygenated form is smaller than normal. For purposes of description one may assume that the rate of circulation and the amount of hæmoglobin in the blood are both normal, although in practice several forms of anoxia may occur in the same patient at the same time.

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When the blood containing less than the normal percentage of oxygenated hæmoglobin reaches the tissues, as much oxygen as possible is removed from it so that the venous blood returning to the lungs also contains less oxyhæmoglobin than usual. Even so, however, the amount of oxygen made available to the tissue is reduced.

Anoxic anoxia occurs as a result of the following causes :—

1. *Obstruction* to respiration which may be,

    (*a*) Mechanical, due to some defect in the apparatus in use.

    (*b*) Anatomical, as a result of some alteration of the form of the natural air passages either from spasm, usually of the larynx or of the intercostal muscles and diaphragm, or from relaxation produced by the anæsthesia allowing tongue or palate to fall into an abnormal position.

    (*c*) Pathological. Injury may produce defects similar to those in (*b*). Disease of the lungs or air passages, pressure from abnormal surrounding structures or the presence of mucous secretion, blood, inhaled material, &c., may all obstruct respiration.

2. *Depression of Respiration,* which may be caused by disease or much more commonly by the action of sedative or anæsthetic drugs themselves.

3. The movements of the lungs may be impaired even though the response of the breath-regulating mechanism is normal and the air-passages clear. This may follow injuries to the chest wall or diaphragm or may be consequent upon the position in which the patient has been placed for operation. It may result from paralysis of respiratory muscles by the anæsthetic drug.

4. Anoxial anoxia may result from the administration of a mixture of gases in which oxygen exerts less than its normal pressure. Generally speaking this condition need only arise during the administration of nitrous oxide and perhaps ethylene.

5. Defects in the anæsthetic machinery in use may, if undetected, lead to inadvertent anoxic anoxia.

The likelihood of periods of anoxic anoxia is as great and perhaps greater during the period of so-called recovery from an anæsthetic as it is during the time in the operating room. The skill of the attention which a patient receives during this period is of the greatest importance and the presence or absence of such skill on the part of the nursing staff may well do more to determine a good recovery and uneventful convalescence than will the actual choice of agent used for the anæsthesia.

B. *Stagnant Anoxia*

In this condition the oxygen-carrying power of the blood and the alveolar pressure of oxygen may be assumed to be normal. The rate at which the tissues receive blood is, however, reduced so that the total amount of oxygen available to them in given time is less than normal. The condition may arise from any disturbance of the circulation which

leads to a reduced cardiac output, but the commonest circumstance in anæsthesia is that where the effective circulating volume of the blood is reduced, as in " secondary " shock.

Two important factors must be remembered in connection with this and the next-to-be-described form of anoxia, anæmic anoxia. First, although the condition is less demonstrative and impressive than anoxic anoxia, similar and equally grave sequelæ may follow a period of stagnant anoxia as may follow acute suffocation.

Second, if stagnant or anæmic anoxia is present and anoxic anoxia be added, obvious florrid clinical signs, such as are usually seen, will not occur. The patient will quietly succumb.

C. *Anæmic Anoxia*

In this condition the alveolar pressure of oxygen, the rate of circulation of the blood and the proportion of the hæmoglobin which leaves the lungs in an oxygenated state may all be assumed to be normal. However, the total amount of hæmoglobin in a given quantity of blood is less than normal so that the amount of oxygenated hæmoglobin, and thus of oxygen, becoming available to the tissues in a unit of time, is less than normal. If anoxic anoxia is added to this condition, cyanosis may not appear because this blue coloration of the body surface depends not upon the proportion of reduced hæmoglobin in the blood, but upon the presence of a certain minimum concentration of it. In an extremely anæmic person it would be possible for all the hæmoglobin to be in a reduced state and yet cyanosis might be absent.

Anæmic anoxia occurs in patients who have suffered hæmorrhage but in whom the blood volume, though not the hæmoglobin concentration, has recovered to normal. It occurs also in anæmias and in carbon-monoxide poisoning where a certain proportion of the hæmoglobin, though present, has been converted into a stable form, carboxyhæmoglobin, and is thus not available for the carriage of oxygen. It is of some interest to note that at different times two anæsthetic agents, cyclopropane and hexobarbitone soluble, have been alleged to have effect similar to carbon monoxide. Careful re-examination of the evidence has however, given assurance that this is not so.

Histotoxic Anoxia

In this form of anoxia, which is typified by the action of cyanide, the aveolar pressure of oxygen, the rate of circulation of the blood and the concentration of oxygenated hæmoglobin in the blood leaving the lungs are all normal. Thus a normal amount of oxygen reaches the tissues but the action of the cyanide prevents, in some way, the tissues from making use of it.

The interest of this matter to anæsthetists is that it has been suggested that all anæsthetic agents have this effect to a lesser and graduated degree and indeed that this constitutes the mechanism whereby they depress

metabolic activity and determine the narcotised state. If this is so, a good deal of our discussion of anoxia might be fruitless because if we are to avoid agents and methods which involve anoxic anoxia, for example, we may only be able to do so by making use of more potent anæsthetic agents. If then these more potent anæsthetic agents all cause histotoxic anoxia we have merely substituted histotoxic for anoxic anoxia and that might not be a desirable exchange.

The evidence for or against this form of anoxia in anæsthesia is both clinical and experimental. In clinical anæsthesia, we may say that, provided that other forms of anoxia are absent, that is to say, the pulmonary ventilation is normal, the uptake and release of oxygen by the blood is normal and the circulation is normal, then we do not see the well-defined sequelæ of anoxia even though these allegedly histotoxic anoxia-producing agents have been used.

The experimental evidence is not easy to evaluate. Experiments with intact animals are of little assistance. Anæsthesia certainly does produce a state of quiescence of muscles and organs, progressively increasing as the depth of anæsthesia increases. This must be associated with decreasing metabolic requirements for oxygen so that observation of such decreases does nothing to prove that it is a deprivation of utilisable oxygen which is responsible for the effect.

Many experiments have been performed to estimate the effect of anæsthetic agents upon the oxygen consumption of isolated tissues. Here we must be careful that it is the *anæsthetic* effect of the agent which is being examined. There is no doubt that ether, or any other potent agent, *can* cause histotoxic anoxia. A man can be killed with ether and that puts an end to oxygen utilisation. In order to be sure that an anæsthetic agent applied to an isolated tissue is acting as an anæsthetic and not perhaps, killing some cells and failing to affect others, one must achieve a reversible effect so that the oxygen consumption of the tissue returns to normal after the removal of the agent. Unfortunately this condition is difficult to fulfil in such experiments even if the anæsthetic were capable of producing reversible histotoxic anoxia. Thus the results of this type of work do not at present allow an unequivocal conclusion.

Recently measurements have been made upon the oxygen content of the blood reaching and leaving the brain of mammals under anæsthesia. Decreased oxygen consumption compared with that in the normal wakened animal has been noted but increasing the depth of anæsthesia did not always further decrease the oxygen consumption and sometimes increased it. One can only say, therefore, that at present there is insufficient evidence for a final conclusion that anæsthetic agents produce reversible histotoxic anoxia of the tissues of the body.

Anoxia due to Decreased Alveolar Carbon Dioxide Pressure

When the pressure of carbon dioxide in the alveolar air and thus in the blood is reduced the oxygenated hæmoglobin less readily gives up its

oxygen to the tissues. Another way of describing this effect is to say that the dissociation curve for blood (fig. 1) is displaced to the left.

During certain types of anæsthesia artificial respiration may be carried out, from choice, for prolonged periods. If there were excessive pulmonary ventilation with reduction of the alveolar carbon-dioxide it is possible that anoxia of this type might result. The effect would probably be small but may be of interest to anæsthetists and worthy of observation.

In conclusion, if we attempt to draw together the various effects of anoxia which have been discussed in terms of clinical language we can hardly do better than cite the list of clinical signs and symptoms of anoxia enumerated by Professor R. M. Waters. They are :—

Acute, Extreme Anoxia

1. Pulse slow and bounding.
2. Pupil dilated, fixed.
3. Respiration depressed or arrested.
4. Systolic pressure raised until respiratory arrest.
5. Muscular convulsions.
6. Cyanosis or pallor.

Moderate Oxygen Want

1. Mental disturbance, anxiety, restlessness.
2. Air hunger.
3. Pre-cordial pain.
4. Vomiting or retching.
5. Pulse rate increased.
6. Systolic pressure slightly raised or depressed.
7. Muscle twitching.
8. Any type disturbance of respiration.

Anæsthetists are naturally more interested in the signs of moderate oxygen want and when we examine them closely it is somewhat disturbing to notice that few of these signs will serve us during deep narcosis. To them may be added the therapeutic test. In this the conditions are temporarily altered so that one can be *sure* that abundant oxygen is available to the patient. *Any* resulting change in the pulse, respiration, colour, &c., indicates that the patient *was* anoxic.

Even with this aid, however, we must admit that while the degree and course of anoxia can be assessed with reasonable assurance in a lightly anæsthetised patient, too little is yet known of the natural history of the disorder to enable us to do this when the anæsthesia is deep. In these circumstances the only safe course is to make sure that the deficiency *cannot* exist. For this we must rely upon the knowledge that there is no respiratory obstruction or embarrassment, that the circulation is adequate, and that we do not administer a mixture deficient in oxygen.

In light anæsthesia we must carefully observe the pulse and respiration; in deep anæsthesia we must " give plenty of oxygen."

GASTRO-INTESTINAL DECOMPRESSION IN THE TREATMENT OF ACUTE OBSTRUCTIONS

Hunterian Lecture delivered at The Royal College of Surgeons of England
' on
15th May, 1947
by

Rodney Smith, M.S., F.R.C.S.
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FOR SOME YEARS now it has been generally realized that the removal by continuous or intermittent aspiration of the foul liquid welling back from the small intestine into the stomach in cases of acute obstruction, can not only materially add to the comfort of the patient, but may also prove a life-saving measure. This " suction drainage," as it has come to be called, of the upper gastro-intestinal tract has now become a standard part of the treatment in both mechanical and functional obstructions. It is my intention to indicate to you why, in my opinion, this form of therapy is of such importance, and to discuss the various types of suction drainage and their application to the different varieties of acute obstruction.

Pathological Basis

A full appreciation of the problem demands an accurate knowlédge of the essential features of the pathology of acute obstruction and it is right that we should begin with a brief consideration of this aspect of the subject.

Essentially, the most important single feature in simple occlusion of the intestine, at whatever site, is the increasing dilatation of the gut and the rising intra-luminal tension above the obstruction. For a while this rising tension merely causes an increase in the rate and amplitude of intestinal peristalsis, but as the tension rises further it begins to exert a harmful effect upon the motility and vascularity of the gut wall. The final result of unrelieved distension is intestinal paresis, both through the direct effect of gross stretching of muscle fibres and also through the indirect effects of local and general circulatory embarrassment and the late results of vascular changes in the gut wall.

I do not propose to enter here into a discussion of the relative importance as lethal factors of loss of fluid from the circulation and toxæmia. I myself believe that although in high obstructions the picture is dominated by loss of fluid and electrolytes, in low obstructions there is in fact a toxæmic factor whose prominence varies with the degree of vascular damage to the gut wall which renders it permeable first to soluble toxins and later even to organisms, both of which normally cannot pass through the intact mucosa. Whether or not you believe that this is so, or whether you believe that loss of fluid is the sole lethal factor, matters little in our discussion to-day. The important point is that distension beyond a certain point can lead to the establishment of a vicious circle, in the presence of

which relief of obstruction alone is insufficient to restore normal intestinal function and save the patient's life. This vicious circle may be represented graphically :
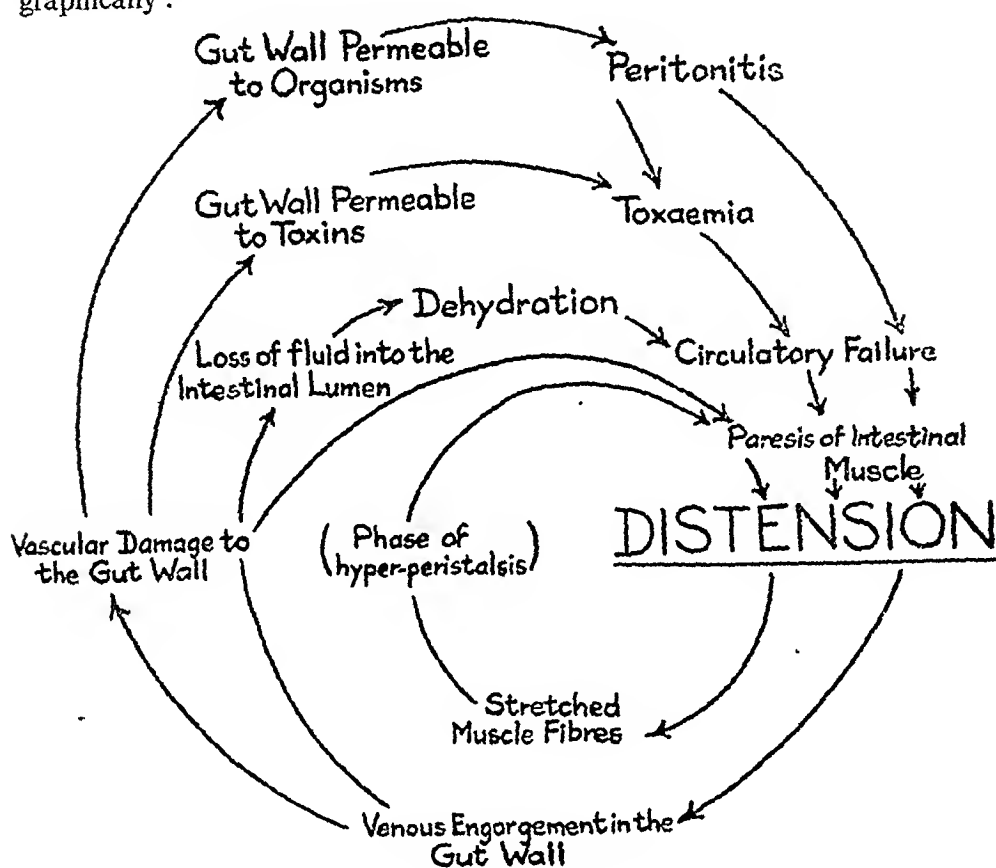
Fig. 1

The early elimination of intestinal distension is thus very desirable, and suction drainage has this as its specific aim, the object being to remove liquid and gas from, and thus decompress, the proximal intestine and to break the vicious circle, or better still to prevent it ever becoming established.

History of Gastro-Intestinal Intubation

The history of gastro-intestinal intubation is interesting. Alexander Munro of Edinburgh[1] intubated cattle with a flexible tube of coiled wire covered with leather as long ago as 1767. A little later John Hunter is believed to have used a stomach tube on human subjects.[2] Aspiration of gastric contents as a therapeutic measure in cases of obstruction dates from 1884[3] and was originally carried out intermittently whenever distension and profuse vomiting indicated the presence of a large volume of liquid in the stomach. Westerman in 1910[4] advocated duodenal intubation, and intermittent aspiration with the tube left *in situ* between aspirations led to the adoption of apparatus for continuous hydrostatic

gastric or duodenal suction drainage through a nasal tube, such as that described by Wangensteen.[5] To the latter and to his collaborator, J. R. Paine, must go much of the credit for the pioneer work on continuous suction drainage.[5, 6, 7]

Later, Miller and Abbott devised a method of intestinal intubation, using a double-lumen tube which was originally designed for physiological studies.[8] The therapeutic application of the Miller-Abbott tube led to the adoption of intestinal suction drainage in certain cases of obstruction, particularly in cases of paralytic ileus, the results of which have been very encouraging.[2, 9, 10, 11, 12, 13, 14]

Even more recently, Harris has described a method of intestinal intubation using a single-lumen tube, the tip of which is surrounded by a thin rubber bag containing mercury.[15] He claims successes in cases normally intubated with a Miller-Abbott tube and believes that the gravitational effect of the mercury bolus is at least as effective in carrying the tip of the tube forward as the air-inflated balloon.

Varieties of Suction Drainage

To-day two main varieties of suction drainage may be recognized :—

(a) Suction drainage with a Levin or Ryle's tube, the tip of which lies in the stomach or duodenum but not beyond—gastric or duodenal suction ;

(b) Intestinal suction drainage with a Miller-Abbott or other tube of similar pattern.

Now at first sight one would imagine that gastric or duodenal suction must be inferior to intestinal suction, and it is true that in certain cases complete decompression can only be achieved by using the intestinal tube. Nevertheless, a more rapid and simple intubation with a Levin or Ryle's tube is sometimes to be preferred, as we shall see.

In cases of obstruction, suction drainage may be employed :—

(a) instead of operation—the Conservative Plan ;

(b) as a pre-operative measure ;

(c) during operation ;

(d) as a post-operative measure.

Let us consider each of these separately.

(a) *Suction Drainage instead of Operation—The Conservative Plan.*

The Conservative Plan of treatment as a substitute for surgery employed in suitable cases undoubtedly represents a major advance in the treatment of acute obstructions, but its application to unsuitable cases can lead to nothing but disaster. It should be considered only if backed by a full appreciation of the indications and contra-indications of the method and of the indications for abandoning it, once started, in favour of surgery. The Conservative Plan may be employed :—

(1) in certain cases of mechanical obstruction of the small intestine ;

(2) in certain cases of functional obstruction. Let us consider these in detail.

The Conservative Plan in mechanical obstructions.

Suction drainage as a substitute for surgery can safely be used in cases of simple occlusion of the small intestine, but its use is only justified if the presence of a strangulating obstruction can be excluded with certainty. If a case of strangulation is treated expectantly in this way considerable clinical improvement may occur owing to the successful decompression of the occluded gut proximal to the strangulated loop, but the illusory nature of the improvement is rudely brought home to the unfortunate clinician when the strangulated loop perforates and peritonitis results. It is here that the real difficulty lies, for there is no sign or symptom or radiological or laboratory investigation which can differentiate easily, and with certainty, a simple occlusion from a strangulating obstruction. Various findings are suggestive but none is conclusive. For that reason, no case of acute intestinal obstruction seen within the first twenty-four hours of the onset of symptoms, should be treated expectantly. If symptoms have been present for more than twenty-four hours the case must be treated on its merits and every effort made to differentiate between a simple and strangulating obstruction. A full account of the diagnosis in acute obstructions would be out of place and take up too much of our time, but this table which summarizes the more important differential points may be of service.[16]

	Simple Occlusion	Strangulating Obstructions	Paralytic Ileus without Peritonitis	Paralytic Ileus due to Peritonitis
Colic	+	+	0	0
Steady pain ..	Late, not severe, only associated with great distension	Severe and appears early	0	+
Tenderness and Rebound tenderness	Late, not severe, only associated with great distension	Severe and appears early	Onset indicates onset of peritonitis	+
Local guarding or rigidity	0	+	0	+
Local mass ..	0	May be present	0	May be present
Peristaltic sounds	+	+	0	0
Change in Leucocyte count	Of doubtful value as a diagnostic aid, but an abnormal count does suggest that the obstruction is not a simple occlusion. Leucopenia sometimes occurs directly after intestinal perforation though it may be due to underlying disease (e.g. typhoid). Leucocytosis is probably an indication not of a strangulating obstruction but of the resultant local peritonitis.			

Fig. 2

To summarize then, the Conservative Plan may be employed in a case of acute mechanical obstruction if :—

(a) the obstruction lies in the small intestine ;

(*b*) acute symptoms have been present for not less than twenty-four hours ;

(*c*) there are no physical or radiological signs suggesting strangulation. In particular, the Conservative Plan should be employed in the very late case of simple occlusion with a grossly distended, dehydrated, toxic and collapsed patient who is in the worst possible state to be subjected to laparotomy. The Conservative Plan has entirely altered the prognosis in such cases. In some cases, for instance, of post-inflammatory adhesions when obstruction has become complete on account of distension, kinking and œdema, proximal decompression actually cures the obstruction and no operation is needed. In others, though proximal decompression is achieved, the obstruction remains and surgery is later required to remedy it. In these cases the Conservative Plan may transform a hazardous, life-or-death intervention of considerable technical difficulty on account of intestinal distension, on a collapsed, moribund patient, into a planned laparotomy, technically much easier, on a patient whose general condition calls for no particular anxiety.

The two essentials of the Conservative Plan are :—

(1) intestinal suction drainage with a Miller-Abbott tube ;

(2) intravenous therapy.

Technique of Intestinal Intubation
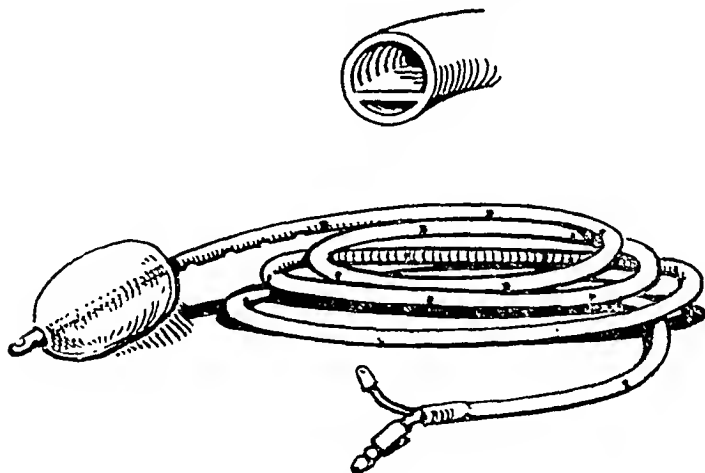
Fig. 3

Many of you will be familiar with the principle of the Miller-Abbott tube. It has a double lumen, one allowing suction to be exerted through a number of holes in the tip, and the other allowing the inflation of a thin rubber balloon encircling the tube just proximal to the tip.

The stomach is first emptied and the tube, with the balloon collapsed, is then passed in exactly the same way as a Levin or Ryle's tube, after which the patient is turned onto the right side so that gravity may lead the tip towards, and finally through the pylorus. Various techniques are

described for assisting the passage of the pylorus and some form of radiological control is essential. A little metallic mercury in the balloon may be of assistance and this stage should never be hurried.

Once the balloon is in the duodenum it is inflated with 20cc. of air, and suction begun. The balloon acts as a bolus and stimulates peristalsis provided that the coil of intestine in which it lies is protected by suction from dilatation. The tip of the tube thus passes down the intestine until it reaches the site of obstruction where it is arrested.

A simple apparatus for providing continuous suction is that originally used by Wangensteen. A modification allows the fluid withdrawn by suction to be collected in a separate vessel as shown here :—

Fig. 4

With the small intestine intubated and suction proceeding, the patient is encouraged to drink, for, with the stomach and small gut emptied, fluid can once more be absorbed if taken by mouth, even with an un-relieved obstruction below, a most important point. Nevertheless, it is unlikely that a sufficient amount of fluid taken by mouth will be absorbed

and, although this can be supplemented by rectal fluids, it is wiser to tie a cannula into the long saphenous vein at the ankle from the beginning. I prefer this to a needle in a vein of the forearm for it is likely that intravenous fluids will be required for several days and for this purpose a cannula is undoubtedly superior. The amount of intravenous fluid thus necessary depends upon the amount of orally-taken fluids which is retained.

It is important to keep a careful fluid balance chart of all patients treated with continuous suction drainage. The chart which I employ is as illustrated here.[16]

1 Date	2 Oral fluid intake	3 Fluid removed by suction	4 Oral balance $(=2-3)$	5 Intra- venous fluid	6 Total fluid $(=4+5)$	7 Urine volume and Sp. Gr.

Fig. 5

The Oral balance, in column 4, is a very important figure, showing how much fluid is being taken by mouth and retained.

Morphine

Indications for morphine. With the suction drainage working and the intravenous drip running, the question of giving morphine arises. This is a most valuable drug but should not lightly be prescribed when the Conservative Plan is being adopted. One of the indications for abandoning the plan altogether in favour of surgical intervention is the onset of signs or symptoms suggesting strangulation. One of these symptoms is local pain ; morphine will unfortunately mask this symptom long enough to place the patient's life in severe danger. A rational plan is this : morphine must be withheld if the cause of the obstruction is not known but only presumed to be a simple occlusion, presumed on account of the length of the history, over twenty-four hours, combined with the absence of any of the signs and symptoms described as suggestive of strangulation. Morphine may be given if the cause of obstruction is definitely known and is not of a type that can lead to strangulation. A therapeutic dose of gr. ¼ should be given and it is unlikely that it will need to be repeated.

Other adjuncts to the Conservative Plan are :—

(i) Oxygen by the B.L.B. mask in cases with gross distension, particularly if shock is present too, continued until the distension is under control ;

(ii) X-ray control. In organic occlusions as in functional obstructions this is essential.

(iii) Prevention or cure of protein deficiency. It has become customary to give one pint of plasma per day during any prolonged period of suction drainage and intravenous fluid replacement to prevent a reduction in the plasma protein level and depletion of the protein stores, which it is now recognized can lead to a breakdown in water balance with œdema, delayed wound healing and other undesirable effects. It is possible that

the administration of plasma will be superseded in time by the administration of protein digests or artificial mixtures of amino acids, which experimentally are more effective in the lasting control of hypoproteinæmia.

Indications for abandoning the Conservative Plan in Simple Occlusions.

(*a*) First and foremost, the Conservative Plan must be abandoned in favour of surgical intervention if it is suspected that an incorrect diagnosis has been made and that a strangulating obstruction is present. The points in the differential diagnosis which are of significance have been sufficiently stressed already.

(*b*) Secondly, operative intervention is necessary if the continuous suction drainage fails to achieve decompression.

(*c*) Thirdly, if continuous suction drainage achieves and maintains decompression but if spontaneous relief of the occlusion does not occur. This is shown by the failure of gas shadows to appear in the colon in check X-rays and the recurrence of clinical and radiological signs of occlusion if suction is stopped.

(*d*) As in functional obstructions, operative intervention becomes necessary if attempts at intestinal intubation end in failure. Exploratory laparotomy should be performed unless the general condition of the patient will not permit this, in which case Intestinal Suction Gastrostomy, an operation which I shall describe in more detail in a moment is preferable to blind enterostomy.

The Conservative Plan in Functional Obstructions.

Clinically, functional obstructions fall into two main groups, those in which paralytic ileus is accompanied by peritonitis and those in which peritonitis is absent, and except in post-operative functional obstructions, it is important to draw this distinction, for the presence or absence of peritonitis determines the treatment. If peritonitis is present, the need for laparotomy is dictated by the fact that the peritonitis is almost certainly secondary to a local inflammatory lesion in the abdomen such as acute appendicitis or a perforation, and although suction drainage plays its part in the treatment of such cases it should not be employed as a substitute for surgery. Paralytic ileus in the absence of peritonitis, however, does not call for urgent surgical intervention and the Conservative Plan is the treatment of choice. The passage of the tube, the details of intravenous therapy, the fluid balance chart, the X-ray control of decompression, and other details of management differ only slightly from those in cases of organic occlusion treated with the Conservative Plan, though the balloon of the Miller-Abbott tube passes more slowly down the small intestine ; but pass it does, even when, before intubation, auscultation detects a completely silent abdomen. As suction empties each coil, the restored muscular tone allows the return of peristalsis and the balloon is passed onwards.

In cases of paralytic ileus treated with the Conservative Plan, morphine is a most valuable drug if given in small repeated doses of gr. $\frac{1}{6}$ to $\frac{1}{8}$, six-hourly.

Indications for operative intervention in Functional Obstructions treated by the Conservative Plan

Two main indications exist for abandoning the Conservative Plan in favour of surgical intervention in functional obstructions. Firstly, if it becomes clear that an incorrect diagnosis has been made and that peritonitis is present. (Again this does not apply to post-operative obstructions.) Secondly, if satisfactory intubation and decompression cannot be achieved. As regards the latter, as one becomes familiar with the technique of intestinal intubation, one's failures become less frequent. There will, however, always remain cases in which the tip of the tube will not pass the pylorus. Unfortunately, these are often the very cases in which intestinal suction is considered essential, those with gross distension, rapid reflux of intestinal fluid into the stomach, dehydration, toxæmia and circulatory failure. The choice in such a case is threefold : one can accept gastric suction as a poor substitute for intestinal suction, and though this adds to the comfort of the patient by keeping his stomach empty and preventing vomiting, it is unlikely to be sufficient in a patient with an established ileus with gross intestinal dilatation ; one can perform an enterostomy and, although we have most of us, at one time or another, seen a moribund patient dramatically saved by this procedure, the mortality of enterostomy in cases of paralytic ileus verges upon 100 per cent. ; thirdly, one can open the abdomen for the purpose of intubating the small intestine with a Miller-Abbott tube. I have twice performed an operation which may be described as Intestinal Suction Gastrostomy in cases in which I considered intestinal suction essential but had failed to intubate in the normal way. Both of these were post-operative cases but the operation may conveniently be described here.

The technique is as follows :

(*a*) A stomach tube is passed, the stomach is emptied and kept empty by suction throughout the operation.

(*b*) A small right upper paramedian incision is made under local anæsthesia, and exposes the pylorus and pyloric antrum (Fig. 6).

(*c*) A stab-incision is made through the left upper rectus muscle and a Miller-Abbott tube, with the balloon collapsed, is threaded through it and drawn out of the main incision (Fig. 7).
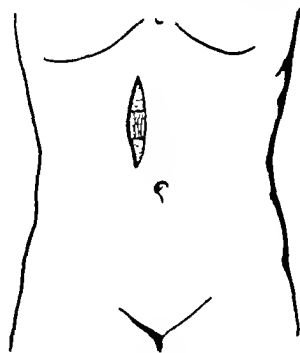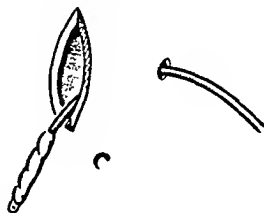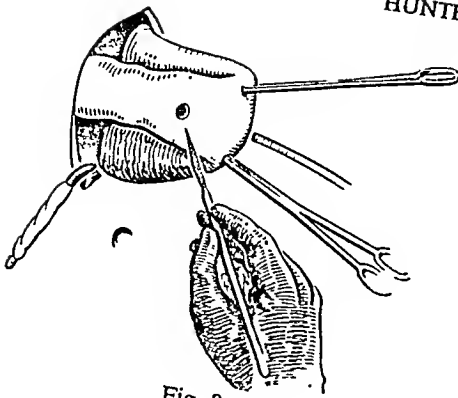

Fig. 6

Fig. 7

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Fig. 8

Fig. 9

Fig. 10

Fig. 11

Fig. 12

Fig. 13

Fig. 14

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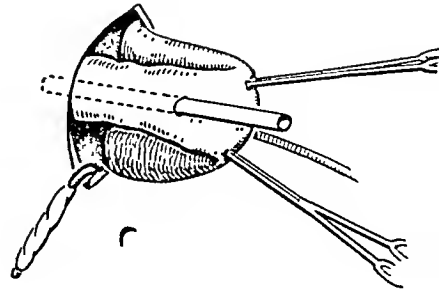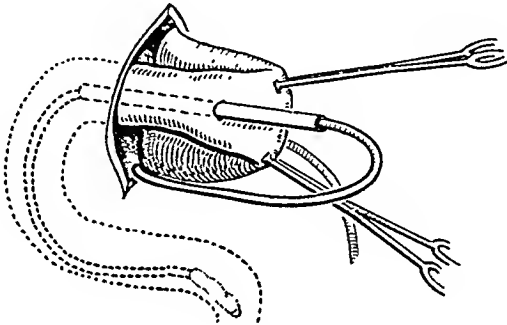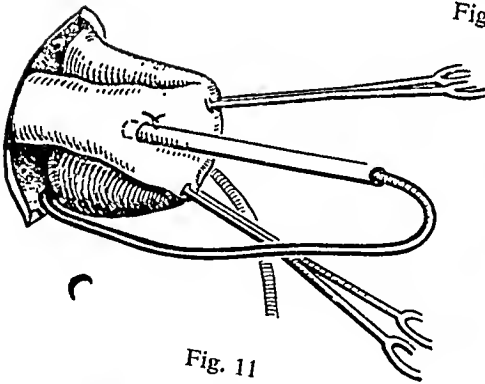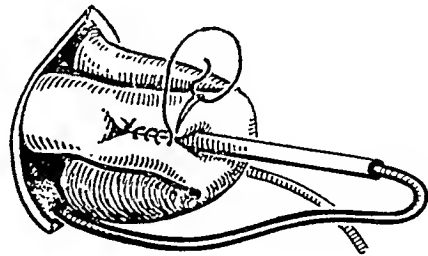
(*d*) The pyloric antrum is drawn into the wound and a stab incision made one inch from the pylorus (Fig. 8).

(*e*) Through this a rigid rubber tube large enough to transmit the Miller-Abbott tube is passed and pushed through the pylorus into the first part of the duodenum (Fig. 9).

(*f*) The Miller-Abbott tube is lubricated and passed through the rigid tube, with sufficient length to carry the tip round to the duodeno-jejunal flexure (Fig. 10).

(*g*) The rigid rubber tube, now ensheathing the Miller-Abbott tube, is withdrawn a sufficient distance to leave its end just inside the stomach, where it is fixed with a single catgut suture. (Fig. 11).

(*h*) Four-fifths of this tube is now " tunnelled " as in performing a Witzel gastrostomy (Fig. 12).

(*i*) The stomach is returned to the abdomen, and the slack of the Miller-Abbott tube inserted through the stab incision is withdrawn until finally the remaining one-fifth of the rigid rubber tube is brought out and anchored to the skin by a single suture (Fig. 13).

(*j*) The main incision is now closed (Fig. 14).

(*k*) The balloon of the Miller-Abbott tube is inflated, continuous suction is applied and intestinal decompression proceeds.

I note, with considerable interest, that John Devine,[18] in his admirable paper on paralytic ileus in the British Journal of Surgery, says that he, in similar cases, has inserted the Miller-Abbott tube directly into the small intestine through a jejunostomy. The principle is, of course, the same, but I believe that the tube should be inserted through a gastrostomy and not a jejunostomy because there is then no risk of narrowing the intestine, closure of the fistula afterwards is speedy and certain, and there is less risk of contaminating the peritoneum with intestinal organisms.

Two questions I have sometimes been asked about this operation should be mentioned. One is, why not pass the tube in the usual way into the stomach and, opening the abdomen, manipulate the tube through the pylorus, palpating the tube through the stomach wall without opening it? The answer is that although this is perfectly possible and has often been done at the end of an exploratory laparotomy, it is much harder to perform than one would imagine and it requires an incision which will admit the whole hand of the surgeon, whereas intestinal suction gastrostomy can be performed through a small incision under local anæsthesia, rapidly, with certainty and with minimal manipulation of the viscera. The other question is, why the rigid tube around the Miller-Abbott tube? The answer is that firstly it is easier to intubate the duodenum with a rigid tube, and secondly that it protects the Miller-Abbott tube from kinking as the patient sits up and moves about in bed, a factor which Vaughan Hudson found troublesome in cases he treated as early as 1941 with post-operative gastric suction through a gastrostomy.[19]

If intestinal suction gastrostomy is performed, the details of management do not differ from those in cases intubated in the normal manner. One last point about the Conservative Plan, and a very important one. Frequent re-examination of each case is essential. In fact, unless the surgeon is prepared to re-examine his patient at very frequent intervals, especially during the first twenty-four hours, he had better not employ the Conservative Plan at all.

(b) Pre-operative Suction Drainage.

So far we have considered suction drainage employed with the intention of avoiding surgery altogether if possible. Now what of the case in which the Conservative Plan is contra-indicated, in which on account of suspected strangulation or other cause early laparotomy is considered necessary. In my opinion pre-operative gastric intubation should never be omitted. If a patient with acute obstruction is allowed to come to the operating theatre with a stomach full of foul intestinal contents, there is a considerable risk of vomiting during the induction of anæsthesia, and the vomit is apt to be sudden and copious. Aspiration into the air passages may easily occur. For a long time now this risk has been recognized and guarded against by emptying the stomach before operation with a large stomach tube. The adoption of this procedure is a step in the right direction, but it does not go far enough. In acute intestinal obstruction the stomach is constantly filling up from the small intestine, and although it may be emptied once completely, sufficient accumulation of fluid may occur to make vomiting during induction still possible.

The logical procedure is to empty the stomach completely with a large stomach tube and then to pass a Levin or Ryle's tube and apply continuous suction drainage to keep the stomach empty. This should be done as soon as possible after diagnosis of acute obstruction has been made, so as to allow the longest possible pre-operative period of suction drainage. A pre-operative intravenous drip is not necessary in the early obstruction but is essential in the late case with marked dehydration or circulatory failure. The question of how much time should elapse before operation must be decided on the merits of each individual case. In uncomplicated cases one hour allows time for the pre-operative injection to take effect and for the apparatus for suction drainage and the intravenous drip to be set up. Further delay is probably not justified, as what is gained in pre-operative decompression is lost in the increased chance of vascular damage to the gut, should strangulation be present, though the correction of gross dehydration may call for a slightly longer pre-operative interval, in which case re-assessment at hourly intervals should be made and operation undertaken as soon as the operative risk is considered to be less than the risk of gangrene from strangulation if there is further delay.

Operation may also be delayed for a short while in one type of strangulation, fortunately rarely seen in these enlightened days. Occasionally a very ill patient with a strangulated external hernia is admitted with a

history of strangulation for well over twenty-four hours, in some cases for days, and it is quite obvious from local and general signs that gangrene of the strangulated gut has already occurred. Nothing is to be gained by rushing a patient of this kind into the operating theatre directly on admission. The damage is already done, and it is necessary now to repair the results of strangulation, not avert them. Resection of the gangrenous gut is clearly going to be necessary, and to subject a toxic, dehydrated patient with gross intestinal dilation to an immediate laparotomy and resection of gut is only justifiable if either it is impossible to improve the poor condition or if some special risk is attendant upon delay. Neither of these two conditions applies. The poor condition can be improved by continuous suction drainage and intravenous fluids and there is no special risk in leaving a strangulated gangrenous loop in an external hernia for a few hours in order to obtain the maximum benefit. True, infection confined to the herniae sac may, during these few hours, spread through the neck of the sac into the general peritoneal cavity, but this risk is less than that of subjecting a moribund patient to an operation he is unlikely to survive. True also, so long as the gangrenous loop remains, the toxic products of autolysis will be absorbed. Nevertheless, on balance this continued toxæmia is more than compensated for in its effects on the general condition of the patient by elimination of the factor of dehydration and even a partial pre-operative decompression of the proximal gut.

(c) *Suction Drainage during Operation.*

There is every reason for continuing suction drainage during the actual operation. Pre-operative decompression is only partial, except in patients who are treated by suction drainage in preference to surgery and who are successfully decompressed but in whom the organic obstruction is not relieved thereby. The object of the surgeon in every case should be to relieve the obstruction and to achieve and maintain complete decompression of the gut above the site of obstruction until normal intestinal function is resumed. The sooner both of these objectives are attained, the better the prognosis. Without suction drainage continued during the operation the benefits of a pre-operative partial decompression are largely lost, for the moment suction drainage is discontinued the partially decompressed proximal gut starts to dilate again, and by the end of the operation considerable ground may have been lost. Further, if the stomach is emptied pre-operatively with the idea of preventing vomiting and aspiration of vomit during the induction of anæsthesia, and if it is not kept empty during the operation, the same risk exists as the patient reaches a lighter plane of anæsthesia at the end of operation, for the stomach may fill up from the small intestine extremely quickly in cases of obstruction.

(d) *Post-operative Suction Drainage.*

There is, I think, little doubt that post-operative suction drainage has had a profound effect upon the fatality rates in cases of acute obstruction. In my opinion, it has been shown quite conclusively that, except possibly

in the very early case with no proximal intestinal dilatation, a short period of post-operative suction should always be prescribed. The pathological basis is easy to understand. Whatever the type of obstruction, operative relief is not followed by immediate resumption of normal intestinal function. After any laparotomy, a temporary physiological ileus is inevitable and this is likely to be more marked if pre-operative trauma, in the shape of distension of the gut, is present. The gut must be tided over the period of post-operative ileus and not be allowed to become stretched by passive dilatation. If the patient comes to the operating table with a moderate degree of intestinal dilatation and no suction drainage is employed after the obstruction is relieved, the post-operative physiological ileus may cause just that little extra dilatation which results in a vicious circle with paralytic ileus and all the results of vascular damage to the gut wall. If the degree of dilatation is slightly less, an irremediable ileus does not develop, but the patient passes through a troublesome few days with distension, nausea and often vomiting. Post-operative suction drainage alters all this. While peristalsis is in abeyance, the gut is protected from dilatation and undergoes no stretching or vascular damage. There is no abdominal distension, nausea or vomiting, and a much smoother convalescence is experienced. After a resection or anastomosis, suction drainage ensures that there shall be no tension on the suture line. It is not, I think, out of place to comment here on the effect which post-operative suction drainage had upon prognosis in cases of abdominal wounding with intestinal perforations, in which the problems are not, after all, very different from those of intestinal obstruction. By the end of the war it had become an inflexible rule in every theatre of war that these cases must have post-operative suction with an indwelling tube, and I myself have no doubt whatever that the astonishingly low fatality rates during the closing stages of the war were due very largely to the routine adoption of this form of treatment.

Types of Post-operative Suction Drainage

Suction drainage employed post-operatively may again be gastric or duodenal, or intestinal. If at operation it is decided that intestinal suction drainage is the method of choice, the Miller-Abbott tube should be passed into the stomach by the anæsthetist, unless it has already been passed pre-operatively, and the surgeon should manipulate the tip through the pylorus. A few years ago it seemed to me that all cases in which marked intestinal dilatation had been noted at operation, or in which a resection of gut or a lateral anastomosis had been necessary, should be treated by intestinal suction drainage with a Miller-Abbott tube. Latterly, however, I have been struck by the fact that these cases seem to do just as well with gastric suction provided that peristaltic sounds are audible with a stethoscope before operation. This clinical observation agrees with the fact clearly demonstrated by John Devine ([18]) that gastric dilatation initiated by stasis in the upper digestive tract causes peristaltic inhibition while

gastric decompression restores intestinal activity. My practice is now to employ post-operative gastric suction except in those cases in which pre-operative auscultation has identified an already established paralytic ileus, when a Miller-Abbott tube is employed. The gastric suction is abandoned in favour of intestinal intubation should signs of a post-operative ileus appear in spite of it.

Post-operative Intravenous Therapy

Except in the very early case with no dehydration, the post-operative intravenous drip infusion is a necessary complement of suction drainage. The volume and chloride content to be given vary from case to case and depend upon the initial degree of dehydration and the rapidity with which the intestine recovers its absorptive function and motility. As in cases treated with the Conservative Plan, if a prolonged period of suction drainage and intravenous fluids becomes necessary, depletion of protein must be guarded against by transfusions of plasma or possibly by the administration of casein hydrolysate or an artificial mixture of amino acids. Once again, small repeated doses of morphine are valuable in the immediate post-operative period.

When to stop Suction Drainage

How long must post-operative suction continue ? The answer is simple —until the resumption of normal intestinal motility and absorption ensures that passive dilatation of the gut will not occur if suction is withdrawn. Evidence that this stage has been reached is provided by the observations that :—

(a) On auscultation, peristaltic sounds can be heard.

(b) If gastric suction drainage is being employed, the fluid withdrawn by suction no longer contains bile.

(c) The fluid balance chart shows that the oral balance has risen to about 80 per cent. of the oral intake.

(d) Plain X-rays, if taken, show :—

(i) Gas shadows in the large gut.

(ii) No dilatation or fluid levels in the small gut.

If any doubt exists of the wisdom of stopping suction, the tube should be left down, but clipped off. If no clinical or radiological signs of dilatation of the small gut follow, the tube may be removed.

Particular care should be taken not to stop suction too soon in cases complicated by peritonitis or in which a resection of gut or a short-circuit operation has been performed. It is common in a case of this kind for a secondary depression of intestinal function to be shown at about the fifth day by a drop in the oral balance, hitherto rising satisfactorily. It is interesting that the fluid balance chart should show this feature, for in just this kind of case, if suction drainage is not employed, the patient very

often does extremely well for three or four days and then suffers a setback with abdominal distension, nausea and sometimes vomiting. It is highly desirable that the patient should be tided over this critical period by keeping the small gut decompressed with suction drainage and that purgatives and enemata should not be given instead.

The Post-operative Fluid Balance Chart

Besides ensuring that the proper correction of dehydration has been achieved and is being maintained, a carefully kept fluid balance chart can give most valuable information about the motility and absorptive function of the gastro-intestinal tract. A mechanical hold-up or paresis of the gut causes stasis and the consequent withdrawal by suction of a high proportion of the fluid taken by mouth.

A few typical charts in which oral intake and oral balance are shown graphically, illustrate this (16)
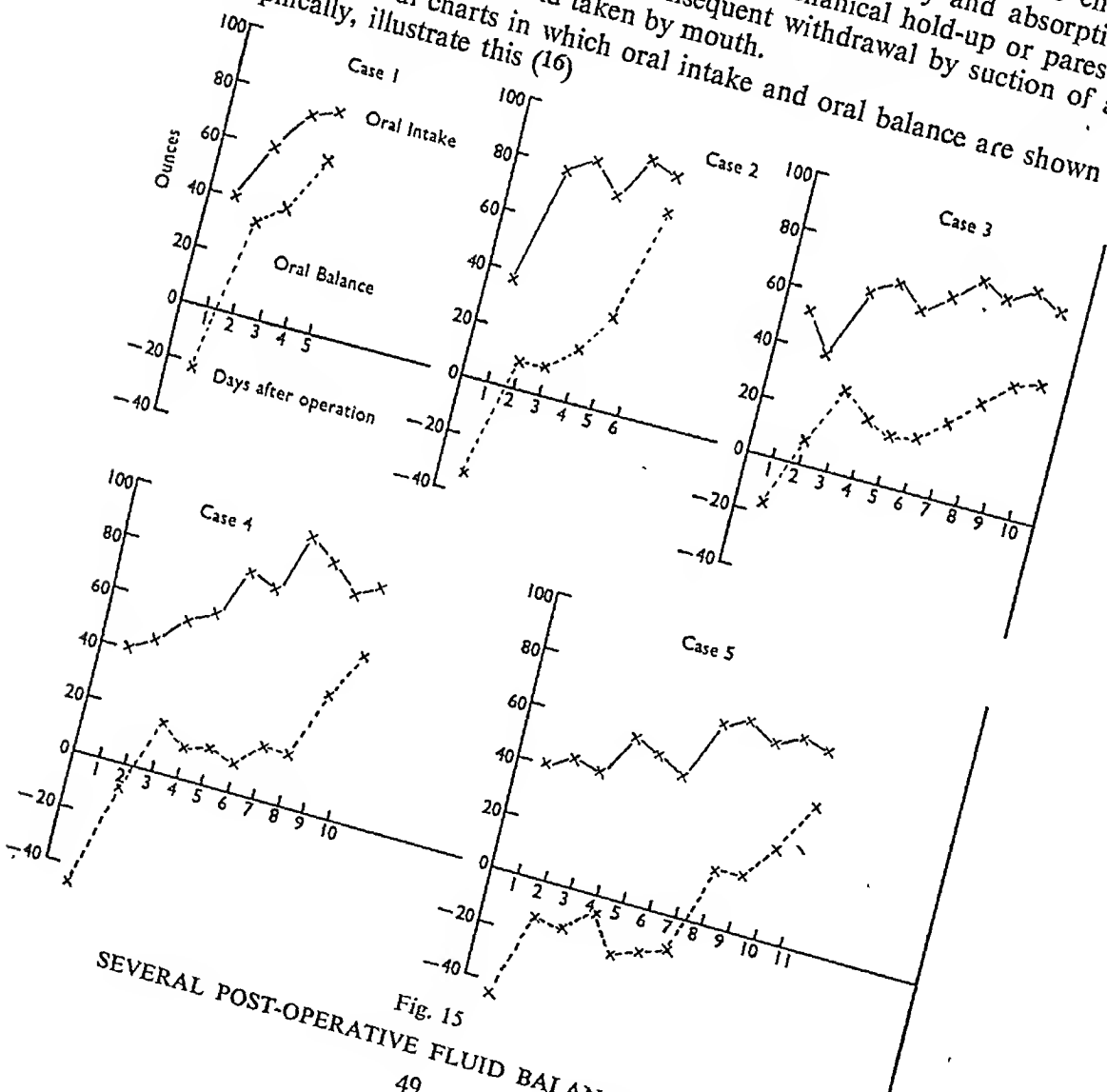
Fig. 15

SEVERAL POST-OPERATIVE FLUID BALANCE CHARTS

CASE 1 Simple release of strangulated inguinal hernia. Uneventful convalescence.

CASE 2 Simple release of strangulated inguinal hernia. Satisfactory convalescence, though rather slower than case 1.

CASE 3 Strangulated femoral hernia. Resection of gangrenous ileum. Slow return to normal intestinal function with a low oral balance from the third to seventh day.

CASE 4 Resection of gangrenous jejunum strangulated by a band. Stormy convalescence with low oral balance until the eighth day.

CASE 5 Simple release of strangulated inguinal hernia. Post-operative paralytic ileus controlled by suction and intravenous therapy. Patient recovered from ileus but died suddenly on the 14th day. Autopsy revealed pulmonary embolism and nil abnormal inside the abdomen.

Post-operative Obstructions

A word on post-operative obstructions. Three varieties may be recognized :—

Firstly, organic obstruction at a single site.

There are many possible causes—for instance œdema at an entero-anastomosis, adhesion and kinking of a coil of intestine, later compression by an inflammatory band. Diagnosis does not differ from that in any other variety of organic obstruction and there is the same choice of treatment, the Conservative Plan or laparotomy. Time does not permit a detailed discussion of each individual type of post-operative focal obstruction and, indeed, this is unnecessary, for the guiding principle is exactly the same as in any other obstruction, namely, that early laparotomy is obligatory if a strangulating obstruction is suspected, but that the Conservative Plan is preferable, at least at the outset, in a non-strangulating obstruction.

Post-operative Functional Obstruction—Paralytic Ileus.

The treatment of post-operative paralytic ileus is conservative, whether or not general peritonitis is present, and it may be observed in passing that a true post-operative ileus is rare in the absence of peritonitis, though it may occur. Laparotomy is pointless unless performed for the purpose of achieving an otherwise impossible intubation of the small intestine, as already described, in order to eradicate some inflammatory or perforative condition developing since the previous laparotomy, such as an extension of a mesenteric thrombosis, or in order to drain a localized collection of pus.

Post-operative Plastic Peritonitis.

Probably the most common cause of post-operative obstruction is plastic peritonitis after, for instance, a very dirty appendicectomy or a resection of a gangrenous loop of intestine. This is a mixed obstruction in which areas of paresis probably exist, but in which the picture is usually dominated by partial organic obstruction at multiple sites from gumming together of coils of intestine with kinking, œdema and compression. It is persistently and incorrectly labelled paralytic ileus, from which it can easily be distinguished clinically by auscultation of the abdomen. The treatment is expectant, suction drainage with a Miller-Abbott tube or failing that a duodenal tube. Laparotomy should be undertaken only to drain a localized abscess.

The Disadvantages and Hazards of Gastro-Intestinal Intubation

I have put before you the very strong arguments for the routine use of suction drainage in cases of intestinal obstruction. Before concluding, I think it is right to consider briefly whether or not there are any outstanding disadvantages or hazards connected with this form of treatment. Firstly, as to disadvantages : it is a fair criticism that it is not pleasant for the patient to suffer an indwelling tube after operation and that most patients would get better without it. I would answer to this that in any attempt to formulate a planned post-operative régime, it is clearly desirable that the interests of the more dangerously ill patients should carry more weight than the fear of adopting an unnecessary line of treatment in less ill patients. In deciding whether to recommend, as a routine, any procedure as a prophylactic against a possible future complication, it is thus beyond doubt better to err on the side of safety and risk over-treating the patient whose life is not in danger rather than risk under-treating a patient whose life may be lost thereby. I am well aware that if routine post-operative suction is employed as an inflexible rule many cases must suffer unnecessary intubation. It would, for instance, be foolish to deny that the vast majority of patients operated on early for a strangulated external hernia get better without suction and, indeed, I have myself frequently omitted post-operative suction in very early cases of this kind for one reason or another. Nevertheless, it cannot be denied that pre-operative suction with a tube left down during operation, even in early cases, is desirable if only from the anæsthetist's point of view, and it is merely one stage further to keep the tube down for another twenty-four, thirty-six or forty-eight hours to tide the patient over the immediate post-operative period.

The necessity for suction drainage in the late case with gross distension or gross vascular damage to the intestine cannot be gainsaid, but among the hazards of intestinal suction drainage with a Miller-Abbott tube must be mentioned the exhaustion of a very ill patient which may sometimes be brought about by a prolonged battle to induce the tube to pass the pylorus, particularly if the battle ends in defeat. It sometimes happens that, faced with a severe post-operative peritonitis and paralytic ileus in a critically ill patient, and realizing that a Miller-Abbott tube in the small intestine would probably solve the problems, one is nevertheless most apprehensive of the effect of the manipulations occasioned by the passage of the tube, particularly if the intubation should turn out to be difficult. Nothing is to be gained by adopting an attitude of " come what may, this tube shall go down." One must preserve a sense of proportion and in each case balance the benefits likely to result from intestinal intubation against what may be a very exhausting ordeal to a patient with perhaps a very slender grasp on life. My practice in such cases is to try the effect of continuous gastric suction drainage first, and see what happens. Quite often

one gets a pleasant surprise, and with the stomach emptied of several pints of foul liquid, deflation of the intestines occurs with the resumption of peristalsis and the passage of flatus. If, nevertheless, the abdomen remains silent and distended, clearly a time comes when somehow or other the intestine must be intubated, if necessary through a gastrostomy under local anæsthesia, as I have described, for the patient to have a chance of survival.

Two rare hazards of an indwelling tube should be mentioned briefly. A few cases have been reported [20] of necrosis of laryngeal cartilages, and very occasionally the tube has led to the late development of an œsophageal stricture.

Here are X-rays of a barium swallow from two such patients :
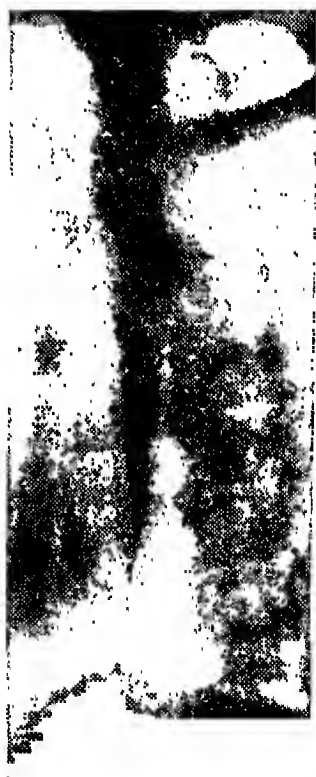
Fig. 16   Fig. 17

Similar cases have been reported elsewhere. The important fact about all these cases is that the illness for which intubation was employed was, in each case, so severe that the patient was in extremis, often for days. The interpretation which suggests itself is that in the presence of profound circulatory depression, the resultant tissue anoxia may be so severe that

the œsophageal mucosa is unable to withstand the pressure of an in-dwelling tube and responds by ulceration, with resultant later formation of a stricture.

The point is really mainly of interest in the differential diagnosis of strictures of the œsophagus, early recognition being important since at that stage dilation is easy. The remote possibility of provoking a late œsophageal stricture should not affect the arguments for or against suction drainage, for it is only the extremely ill patient who runs this risk, and, because he is so ill, the treatment of the immediate emergency must outweigh all other considerations.

Fatality Rates

I would like to end this brief survey of a very important subject by referring to the published fatality rates in cases of acute intestinal obstruction during the last fifty years. By the year 1900, a number of notable surgeons had operated on sufficient cases of acute intestinal obstruction to report fatality rates. In this year von Mikulicz recorded a 51 per cent. mortality in 70 cases and von Bergmann a 77 per cent. mortality in 66 cases.[22] Improvement does not appear to have been rapid, for Scudder in 1909 collected 121 cases of acute obstruction at the Massachusetts General Hospital excluding strangulated hernia and neoplasms, with a fatality rate of 60 per cent.[23]

The next twenty years saw a remarkable fall in the fatality rates of all abdominal operations, and in most acute abdominal lesions a 75 per cent. improvement in figures was seen.

In acute intestinal obstructions, however, the improvement in fatality rates was not nearly so marked, a fact commented on by Burgess [24, 25] in his well-known papers in 1929, mortality remaining in the region of 30 per cent. Why, Burgess asked, at a meeting of the Medical Society of London on February 25, 1929, did patients die of acute obstruction? Echo answered " Why?", and therein lies the reason for the failure to reduce the mortality for acute obstruction in the first thirty years of the century to an extent comparable with other abdominal crises. The patient might get to hospital earlier, diagnosis might be more accurate, surgical technique more sure, but if the essentials of pathology and above all the precise cause of death were not understood fully, how could it be expected that treatment would be accurate except in broad outline? It was known. that obstruction, if it persisted, killed the patient, and operation was therefore undertaken to relieve it. It was not known how it killed the patient or what were the lethal factors in the various types of obstruction, and therefore it was impossible to plan a line of action specifically aimed at counteracting the lethal secondary effects of obstruction at the same time as relieving the obstruction itself.

In the early nineteen-thirties Wangensteen and his collaborators showed us the way, and since then the wider adoption of suction drainage and technical advances in the apparatus available have brought about a

phenomenal fall in mortality. Fatality rates of 10-15 per cent. are being reported in large series, in some cases even below 10 per cent. Other factors have of course had their effect in this satisfactory improvement in prognosis. I would mention particularly :—

(1) Improved methods of diagnosis, in particular the use of plain X-rays as an aid to clinical diagnosis ;

(2) Greatly improved methods of combating shock and dehydration by intravenous therapy, improved anæsthetic technique, including the wider use of local anæsthesia, and lately the introduction of Curare and Myanesin ; thus lowered operative risk ;

(3) The realization that peritonitis is a very real danger even in the absence of frank gangrene and perforation ; advances in general and intra-peritoneal chemo-therapy ; the scrupulous avoidance of peritoneal soiling if the gut is perforce opened at operation ; the development of an aseptic technique of resection and anastomosis.

Taking all this into account, however, I would unhesitatingly say that the routine adoption of intestinal decompression has been far and away the greatest single factor in producing the rapid drop in fatality rates in acute intestinal obstruction which the last fifteen years have seen and that a full and complete understanding of all that can be achieved by this most potent therapeutic weapon is an essential possession of any surgeon who hopes to grapple successfully with this most dangerous acute abdominal emergency.

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DIARY FOR JANUARY (15th-30th)

Thur. 15 5.00 Mr. V. H. Ellis—Pyogenic Affections of Hip and Knee Joints.
           5.00 Prof. H. A. Harris—Bone Growth.
           6.15 Dr. E. A. Pask—Physiology of Anæsthesia.
Fri.   16 5.00 Mr. A. Rocyn Jones—Congenital Dislocation of the Hip.
           5.00 Dr. J. Short—Development of the Face.
           6.15 Dr. G. N. Jenkins—Saliva.
Mon. 19 5.00 Mr. F. W. Holdsworth—Fractures Involving the Knee Joint.
           5.00 Dr. J. Whillis—Walls of the Oral Cavity.
           6.15 Prof. H. F. Humphreys—Tooth Form and Function.
Tues. 20 5.00 Mr. E. P. Brockman—Congenital Deformities of the Foot.
           5.00 Dr. J. Whillis—Deglutition.
           6.15 Dr. W. V. Thorpe—Calcification.
Wed. 21   Primary F.R.C.S. Examination begins.
           5.00 Mr. R. Broomhead—Muscle and Tendon Ruptures.
           5.00 Mr. G. Heaton—Fluorosis.
           6.15 Prof. F. Goldby—Trigeminal Nerve.
Thur. 22 5.00 Mr. P. Wiles—Postural Deformities of the Spine.
           5.00 Prof. R. J. Brocklehurst—Taste.
           6.15 Mr. A. Bulleid—Oral Bacteriology.
Fri.   23   Final L.D.S. Examination begins.
           5.00 Mr. A. J. Watson—Ankle Joint Fractures.
           5.00 Prof. F. C. Wilkinson—Focal Infection.
           6.15 Miss A. Shore—Proteins.
Mon. 26 5.00 Mr. H. J. Burrows—Static Derangements of the Foot.
           5.00 Prof. J. F. D. Shrewsbury—The Modes of Transmission of Bacterial
                 Diseases, with special reference to the Transmission of Oral
                 Infections (Part I.)
           6.15 Mr. E. B. Manley—The Structure of Human Enamel.
Tues. 27 5.00 Mr. R. Barnes—Brachial Flexus Injuries.
           5.00 Prof. J. F. D. Shrewsbury—The Modes of Transmission of Bacterial
                 Diseases (Part II).
           6.15 Mr. E. B. Manley—Dentine and Pulp and Their Functions.
Wed. 28 5.00 Mr. R. B. Young—Derangements of Lumbar Spine and Pelvic Joints.
           5.00 Prof. W. T. Astbury—Collagen and Keratin.
           6.15 Mr. E. B. Manley—The Role of the Epithelium in Tooth Develop-
                 ment.
Thur. 29 5.00 Mr. St. J. D. Buxton—Fractures and Dislocation of the Shoulder
                 Joint.
           5.00 Prof. R. V. Bradlaw—Tumours of the Jaws.
Fri.   30 5.00 Mr. G. B. Pritchard—Pathology of Pulp.

DIARY FOR FEBRUARY

Mon.   2 5.00 Prof. A. W. Badenoch—Hunterian Lecture—Congenital Obstruction
                 at the Bladder Neck.*
           5.00 Dr. C. C. N. Vass—Alimentary Tract (Part I).
           6.15 Prof. M. A. Rushton—Parodontal Disease.
Tues.   3 5.00 Prof. A. D. Beattie—Hunterian Lecture—Treatment of Peptic Ulcer
                 by Vagotomy.*
           5.00 Dr. C. C. N. Vass—Alimentary Tract (Part II).
           6.15 Dr. E. W. Fish—Connective Tissue.
Wed.   4 5.00 Prof. D. J. Browne—Hunterian Lecture—Hare Lip and Cleft Palate.*
           5.00 Prof. M. S. Lucas Keene—Pathways for Pain and Taste.
           6.15 Prof. D. T. Harris—Pain.

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* Not part of courses.

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Thur.	5	5.00	DR. A. B. McGREGOR—Lymphatic Drainage.
Fri.	6	5.00	PROF. R. G. PULVERTAFT—Hunterian Lecture—Repair of Tendon Injuries in the Hand.*
		5.00	PROF. S. L. BAKER—Reactions of Bone to Injury.
Mon.	9	5.00	PROF. V. H. RIDDELL—Hunterian Lecture—Carcinoma of the Breast.*
		5.00	MR. RAINSFORD MOWLEM—Bone Grafts to the Jaws.
		6.15	MR. A. W. MOORE—Acute Infections arising from Teeth.
Tues.	10	5.00	PROF. F. E. STOCK—Hunterian Lecture—Surgical Approach to Hypertension.*
		5.00	PROF. H. H. STONES—Dental Caries (Part I).
		6.15	SIR STANFORD CADE—Cancer of the Mouth.
Wed.	11	5.00	PROF. A. H. VISICK—Hunterian Lecture—A Study of the Failures after Gastrectomy.*
		5.00	PROF. H. H. STONES—Dental Caries (Part II).
		6.15	PROF. TALMAGE READ—Cysts.
		7.00	Monthly Dinner for Fellows, Members and Licentiates.
Thur.	12	5.00	MR. RAINSFORD MOWLEM—Bone Cartilage and Fascia Transplants.
		5.00	MR. A. D. MARSTON—Anæsthetics in Relation to Dental Surgery.
		6.15	DR. C. H. TONGE—Surgical Anatomy.
Fri.	13		D.O.M.S. Examination (Part II) begins.
		5.00	PROF. S. A. WAY—Hunterian Lecture—The Anatomy of the Lym-• phatic Drainage of the Vulva and its Influence on the Radical Operation for Carcinoma.*
		5.00	MR. G. LEATHERMAN—Oral Hygiene.
		6.15	MR. G. F. ROWBOTHAM—Head Injuries.
Mon.	16	5.00	SIR HAROLD GILLIES—Skin Flaps : Indications and Technique.
		5.00	DR. E. W. FISH—Gingivectomy.
		6.15	DR. J. F. BROMLEY—Radiotherapy in Tumours of the Mouth.
Tues.	17	5.00	PROF. M. C. WILKINSON—Hunterian Lecture—Observations on the Pathogenesis and Treatment of Skeletal Tuberculosis.*
		5.00	MR. T. WARD—Surgery in Relation to Prosthesis.
Wed.	18	5.00	MR. A. B. WALLACE—Free Skin Grafting—Methods and Application.
		5.00	SIR ALEXANDER FLEMING—Penicillin.
		6.15	PROF. H. STOBIE—Role of Dental Surgery in Medicine.
Thur.	19	5.00	PROF. J. BEATTIE—Hormonal changes after Injuries.
		5.00	MR. S. H. WASS—Osteomyolitis of Mandible.
		6.15	DR. S. BLACKMAN—Radiology.
Fri.	20	5.00	MR. R. P. OSBORNE—Burns and Their Early Treatment.
		5.00	MR. H. T. ROPER HALL—Therapeutics in Dentistry.
Mon.	23	5.00	PROF. T. POMFRET KILNER—Cleft Lip and Palate Repair.
		5.00	MR. P. R. SHEPHERD—Fractures of the Mandible (I).
		6.15	MR. F. C. ORMEROD—Diseases of Maxillary Sinuses.
Tues.	24	5.00	MR. P. H. JAYES—Fractures of the Facial Skeleton.
		5.00	MR. P. R. SHEPHERD—Fractures of the Mandible (II).
		6.15	MR. A. McLEOD—Posterior Restorations (I).
Wed.	25	5.00	MR. J. N. BARRON—Hand Injuries.
		5.00	MR. V. ZACHARY COPE—Actinomycosis.
		6.15	MR. B. W. FICKLING—Replacement of Tissue Loss in Jaws.
Thur.	26	5.00	MR. J. B. CUTHBERT—Hand Deformities.
		5.00	PROF. F. H. BENTLEY—The Uses and Abuses of Drugs in Surgery.
		6.15	MR. W. KELSEY FRY—Impacted Third Molar.
Fri.	27	5.00	SIR ARCHIBALD McINDOE—External Genitalia : Treatment of Congenital Deformities.
		5.00	SIR CECIL WAKELEY—Mandibular Joint.
		6.15	MR. H. L. HARDWICK—Gingivitis of Pregnancy.

* Not part of courses.

SIR WILLIAM H. COLLINS

THE ROYAL COLLEGE OF SURGEONS mourns the loss of Sir William H. Collins, Honorary Fellow, Honorary Medallist, Hunterian Trustee and the greatest benefactor in its history. Sir William Collins had the highest regard for the achievements of surgery for he realised that his own life had been saved by treatment based on careful scientific work, and the accumulated knowledge and skill of many generations. He had to submit to operation for phlegmonous appendicitis which was followed by ileus requiring a complete ileostomy for its relief, and eventually had a third operation for the closure of the artificial opening.

After his recovery Sir William Collins turned his mind to how he could help the advancement of medical science and how he could bring the resulting benefits to his fellow men by direct contributions to hospital services. He had already been in the habit of giving generously to hospitals near the sites of his factories, but now sought a wider field for his benefactions. His first outstanding gift was to The Middlesex Hospital where he built the X-Ray Diagnosis Department at a cost of about £50,000, and, on its completion, provided for its endowment by the gift of a further £50,000. The needs of the King Edward VII Hospital at Windsor, near his home at Wexham Park, next attracted his benevolence and he made many munificent gifts both before and after he was elected Chairman.

But Sir William may be said to have found his " spiritual home " at the Royal College of Surgeons. Soon after he became acquainted with the needs of the College he realised its enormous opportunities in research and post-graduate education. He also saw the importance of acquiring room for expansion, and his first gift of £100,000 for the endowment of a Professorship of Human and Comparative Pathology was linked with a recommendation that the Council should purchase the bombed sites to the east of the College. This gift was followed by a further £100,000 for the endowment of a Chair of Human and Comparative Anatomy, and yet a third £100,000 for the general endowment of scientific work.

Wherever Sir William gave financial aid he was also willing to give service. At the College, where he held official status as a Hunterian Trustee, he attended all functions and made himself acquainted with the work of the various departments. He rejoiced in the activity which resulted from his gifts. He visited the College on practically every Council day and the members of the Council and scientific staff soon came to regard him as a personal friend and began to address him by his familiar nickname of " Wilkie." Thus he is mourned in the College as a friend as well as a colleague and benefactor. The Council are happy in the knowledge that they awarded him the highest honours they had to bestow, for " a single flower in a man's button-hole is worth a ton of roses piled upon his
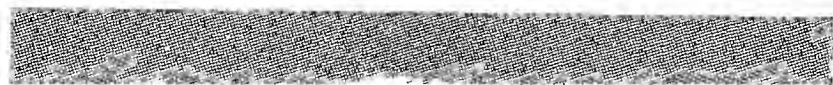
SIR WILLIAM H. COLLINS

grave." They rejoiced when he received Honour from The King. Thousands of his fellow men of this and succeeding generations may thank God for the life and work of Sir William H. Collins.

ADDRESS AT THE MEMORIAL SERVICE IN THE CHAPEL OF THE MIDDLESEX HOSPITAL BY ALFRED PEMBERTON

" We are gathered here to-day to remember, and to pay tribute to the life and work of William Henry Collins. Everyone of us, I think, likes to recall those who are gone, those whom we have loved, in some favourite setting ; in some particular role ; and it is in the role of good companion that I, amongst a great many others, will like to recall with gratitude the friendship and many endearing qualities of Wilkie Collins.

Whether he greeted you as you crossed the threshold of his homes or climbed aboard his beloved yachts on the sea he loved so well, or whether he hailed you at some chance encounter in foreign lands, the genuine warmth of his welcome together with his geniality and charm, never varied. He was always the same.

I think the reason for the exceptional quality of his friendship was based on his intense interest in others—an interest which, owing perhaps to the austere Victorian upbringing of his early days, and the exacting task of business-building, had little chance to manifest itself to the outer world until just over twenty years ago. Suddenly he was faced with a desperately critical illness, but it was God's will that he be brought back to health through the most devoted care and outstanding skill of the medical profession. It was then perhaps that his interest in the things of the spirit quickened. It was then that he made his big resolution ; that he would repay; that he would do all in his power to help those who fight the never-ending war against the sufferings of the human body. This resolution gave him, I think, a fuller life with qualities of sympathy and understanding which had been previously half hidden in the bustle of his busy existence.

A delight in and devotion to children became more evident, (he was always fond of children), and that this devotion came from the bottom of his heart, was evident by the way that children, who are quick to recognise their real friends, returned that devotion with a really genuine affection.

In many mortals, resolutions are apt to weaken with the passing of the years, but strong and steadfast, he gave of the very essence of charity, not only of his worldly goods, lavishly, but of his time, talents and energy, never sparing himself, and always with a jest on his lips. He loved it all, particularly his days at the Royal College of Surgeons.

Those who minister to suffering humankind, and we his friends, will remember him as our good companion and our good friend."

ON CANCER OF THE BREAST

by

Sir Gordon Gordon-Taylor, K.B.E., C.B., LL.D., F.R.C.S.

Being the Opening Address at the Section of Surgery, the Royal Society of Medicine

on

3rd December, 1947

I AM HONOURED in being invited to open this symposium on the treatment of breast cancer. I know not whether I have been chosen as one *maturusne senex*, a sort of ' elder statesman,' or whether the suggestion of my name was prompted by my long association with an institution in which, as far back as 1792, a ward was specially allocated for the study and treatment of cancer patients, a general hospital which has for more than 150 years enjoyed a cancer reputation among the community, which has at all times attracted disproportionate numbers of cancer patients within its precincts, which has numbered on its staff colleagues of my own and of an earlier century who have made noteworthy contributions to our know-ledge and our treatment of mammary carcinoma, and which to-day is possessed of a radiotherapeutic wing and wards under a Director with professorial status.

The story of cancer of the breast is as old as womankind. It finds mention in the " Edwin Smith Surgical Papyrus " dating back to the Pyramid Age, 3,000 years before the birth of Christ, perchance written by the first known physician, the Egyptian Imhotep. Throughout the centuries the malady has respected neither person, age nor clime, and has dealt doom to those of rank and riches as well as to the lowly and the meek. Possibly Queen Atossa, spouse and bedfellow of Darius Hystaspis and of others, as Herodotus tells us, may have suffered from a mammary growth but was cured by the Crotoniat physician Democedes, while Justinian's wife, the famous Empress Theodora, who, if Procopius is to be believed, ascended from the brothel to the purple, perished from the same disease.

Mythology relates the Amazonian practice of burning off the right breast of the girls so that the latter could better handle the bow, a habit which long antedated the tortures of St. Agatha and the cauterizations and diathermy-ablations of the cancerous mamma in more modern times. And in respect of literature who is there that has not been moved by the story of the breast operation in " Rab and his Friends " contained in Dr. John Brown's " Horæ Subsecivæ "?

Time is lacking to recount the whole story of the evolution of what, till a few years ago, might have been veritably termed radical mastectomy performed in the " proper cancer spirit," but I would refer those interested to the learned papers of William A. Cooper[1] of New York and our own lamented Sir D'Arcy Power.[2] The descriptions of many of these pristine operations are reminiscent of the martyrdom of the saints, while the barbarous armamentarium of men like Scultetus, Fabricius Hildanus, Bidloo or Tabor to facilitate the removal of a breast recalls the crude methods and cutlery of *les sales Boches* within more recent times.

The battle round the axillary glands has raged for centuries; by some they have been conveniently ignored, some like the great " Jamie " Syme of Edinburgh and distinguished surgeons of St. Bartholomews in the 'eighties and 'nineties were wont to shrug their shoulders or shake their heads—mistakenly as we know now—when glands could be felt, whereas others from the time of Severinus and Fabricius Hildanus onwards have resolutely urged their removal, even when they were not palpable.

The attack on the cancerous mamma itself has varied with the years from the carnivorous extraction of a tumour, the primitive removal, or alas : the panther-like avulsion of a breast to the more scientific ablation of the whole organ with free and fierce flayings of skin and pectoral fascia and muscle. The spirit of Chauvinism is not yet dead within me, and I gladly pay my humble tribute to those from The Middlesex Hospital who have contributed to the evolution of the radical operation, especially Charles Moore,[3] whose epoch-making paper appeared in 1867, my old surgical teacher Sir Alfred Pearce Gould,* whose private assistant I was for nearly 10 years, and my distinguished colleague and firm friend, W. Sampson Handley.[4] In 1903 as a fellow-worker in our Cancer Research Laboratories at " Middlesex," I saw the inception of the pathological work which has made Handley world-famous, and the operation which he designed in those far-off days to meet pathological requirements has been that on which I have largely modelled my own surgery of mammary cancer.

I may not have followed Handley in his employment of radium at the time of operation or in the use of post-operative radiation, but have always allowed my own enthusiasm the fullest scope and more than a dozen times have deliberately removed the chain of anterior mediastinal glands along with the internal mammary vessels after resection of the second and third costal cartilages. Now in retrospect, I rather regret that I have not availed myself of this added step towards the truly " radical " on many more occasions : my end-results might have been bettered thereby.

Other important contributions germane to this discussion have emanated in more recent times from my own Hospital and are deserving of attention : those of Patey[5], Scarff [5, 6] and R. S. Handley[6] on the histological grading of mammary tumours and its prognostic importance ; Brian Truscott's[7] recent and most valuable paper on a " follow-up " of 1,211 cases of mammary carcinoma treated *omni modo* in "Middlesex" from 1926 to 1940 and Thackray and R. S. Handley[8]—distinguished son of a famous father—who have investigated the anterior mediastinal glands in a series of cases of breast cancer.

This brief historical epitome of the treatment of cancer of the breast might be termed " the wax and wane " of radical mastectomy. For be

* " Moore's suggestions have been the basis of all recent operative procedures." Thomas Bryant, 1902.
Considerable comment was evoked in medical journals in the end of the century anent our British habit of ascribing any new procedure to the foreigner. Bryant speaks of the radical operation as Moore's, Banks', Gould's or Halsted's.

assured, when once the ancillary methods of surgery are added to the knife in dealing with cancer, the extent of the operative removal irresistibly, inevitably becomes curtailed : nor is it otherwise in the treatment of mammary malignant disease. Confidence in pre-operative and post-operative irradiation has for some years indubitably tended to restrict the extent of surgical ablation ; curtailment of operation is noted especially in the amount of skin and muscle removed and the clearance of the axilla. By some, radium has even been regarded as the equal, if not the superior of radical surgical removal !

In order to secure some information as to the practice of other surgeons in respect of supplementary radiation, in 1938, I approached 72 of my surgical friends throughout the length and breadth of Great Britain. Four different usages apparently prevailed a decade ago ; of the surgeons interrogated, 25 per cent. made no use of irradiation as a supplement to radical operation ; 44 per cent. employed some form of irradiation as a supplement to every radical operation ; 29 per cent. used it only after radical operation in Group II cases ; two per cent. favoured irradiation with or without a local operation.

How has the employment of ancillary radiation fared since 1938 ? The distraction of surgical attention towards other problems, especially trauma, during the war years and the absence abroad of many operators of first rank, necessarily thrust much of the treatment of cancer on to hard-pressed but ever-willing radiotherapeutic clinics, perhaps especially in those parts of the country which were happily spared the ravages and disorderings of war. Unfortunately, recommendations are nowadays finding their way into the literature which, so far as the incompleteness of radical mastectomy is concerned, take us back to the procedures of Scultetus and others of the Dark and Middle Ages ! My distinguished and gallant surgical friend from St. Thomas's[9] has no need now to super-scribe above his name the famous passage from Isaiah, for his " wilder-ness " has become a modern " paradise enow."

I am not, and have never been, a "cancer specialist" ; I have never had a cancer clinic or sat among the mighty in Cancer Campaigns. I am a simple soul, a simple surgeon profoundly ignorant of the recondite mysteries of radiation therapy, and very fearful of the wondrous apparatus under the management of these scientific experts. But I would hasten to disabuse you of any thought that I harbour even the very faintest soupçon of antagonism towards my radiotherapeutic confrères, least of all towards my friend and colleague Brian Windeyer, who has so often brought aid to myself and healing and comfort to my patients. I yield to none in my admiration for those who practise the radiotherapeutic art, realising full well the constant demands on their sympathy and cheerfulness, full of wondering amazement at their devotion in a type of work which may well undermine their own health and may often nigh break their spirit. I am cognizant of their wonderful healing of bone metastases, the melting

away of enlarged superclavicular glands and of cutaneous nodules in those with mammary cancer. In respect of the advanced inoperable cases, my debt to these men in whose own breasts there wells the very milk of human kindness is incalculable, and I marvel at their unfailing humanity and enthusiasm in their dealings with a class of patient that is, for the most part, condemned to die. It is for this advanced class of case, or for patients deemed unsuitable for radical surgery on other grounds than the state of the malady that I reserve X-Ray therapy. Save for one very brief and perhaps fortuitously unhappy period long ago, I have personally eschewed the methods of post-operative radiation as a supplement to radical surgery in the cases of mammary carcinoma which are by common usage now classified as Group I and Group II. For such I have preferred a sharp knife, a stout heart and unquenchable optimism, and have regarded the widest radical surgery untrammelled by ancillary radiation as the method of election in almost every case belonging to these two categories. So far as my treatment of cancer of the breast is concerned, I admit that I may be not unfairly likened to him in the Scriptures who received the one talent and who went and digged in the earth and hid his Lord's money, receiving a reprimand because of his lack of enterprise, for despite a surgical stewardship of 40 years in a modern teaching hospital I have just bucolically swept wider and wider with my scalpel and have refrained from temptations which X-Ray experts seductively place before the surgeon.

Nevertheless, I have endeavoured to orientate myself amid the welter of different and varied combinations of surgery and radiation, for I would have been guilty of intransigent conservativism and therapeutic myopia had I not endeavoured to make myself conversant with the claims of others treating primary mammary carcinoma by methods dissimilar to my own. *C'est très difficile ! Quot homines, tot sententiæ.* Thus Regaud of the Fondation Curie wrote that post-operative X-Ray therapy should not be used as a routine, but should be reserved for certain areas specifically under suspicion or for actual recurrence. Other distinguished clinics and clinicians would commence post-operative radiation within a few days of operation, thus, Pfahler[13] in 10 to 14 days, Adair[12] in four to six weeks, etc.; Wintz[14] on the other hand preferred to allow a period of six to eight months to elapse before commencing X-Ray treatment, except in the event of development of nodules or glandular metastases in the meantime. Some savants advise pre-operative radiation, some post-operative, others would also sow the operation area with radium tubes. There is little unanimity concerning technique among the great ones !

A few surgeons such as Haagensen and Stout[11] have dared to confess their disappointment with radiation. Stuart Harrington[10] of the Mayo Clinic, on the basis of a very large experience, found that no matter if axillary gland involvement was present or not, no appreciable difference existed in the percentage survival at 10 years, whether radiation was employed

or omitted. Truscott in his recent investigation thinks that the treatment of cancer of the breast from 1926-35 was best when surgery alone was employed, and from 1935 onwards " there is insufficient evidence to say whether the combination of radiotherapy and surgery is better than surgery alone."

On the other hand, few of the papers dealing with radiation have made any reference to the *morbidity from radiation therapy*: apart from unhappy local effects, the sickness, misery and other untoward sequelæ *may lower the patient's resistance* to any malignant cells that may remain after surgical operation.

Cancer Resistance or Immunity

It is amazing how the disease may return many years after operation, at a time when the original malady has been almost forgotten. Truscott in 1947 from his investigation reiterates the statement of Mitchell Banks in 1900, that no matter how early the case or thorough the treatment, no patient is free from the possibility of recurrence until death occurs from some other cause. Recurrence has certainly taken place as long as 32 years after the initial operation ; Frank Steward[15] recorded a 31 year recurrence in a woman operated on originally by Butlin ; Bryant[16] relates recurrences at 32, 31 and 25 years. Verneuil[18] operated on a recurrence 30 years after the first operation ; Bowlby[17] on a case of Sir Thomas Smith operated on 24 years previously.

I have myself seen a carcinoma of the breast recur in the pelvis as a mass, the histological structure of which was reported as a " spheroidal cell carcinoma of mammary type " : the breast had been radically removed by Sir Cuthbert Wallace 30 years before. Three cases of recurrence in the scar of a radical mastectomy were observed by me 21, 22 and 23 years after operations by other surgeons.

In some cases no causal factor except perhaps *age* can be impugned as bearing any relationship to the recrudescence of the malady, but in others recurrence does appear to have been *preceded by intercurrent disease, or by surgical operation for some independent condition*. In one patient of my own, a recurrence in the scar developed 17 years after a radical mastectomy, and a few weeks after a very severe attack of acute pyelonephritis. I have also observed recurrence in the scar following an operation for hæmorrhoids performed 16 years after the initial amputation. In three cases recurrence followed shortly after a gallstone operation, and in another patient recurrence suggested a relationship to a herniorraphy performed under local anæsthesia ! In such cases, what Sir Alfred Pearce Gould[19] in his Bradshaw Lecture, termed " cancer immunity " appears to have been broken down by happenings unconnected with the original disease. Truscott in his review pessimistically draws attention to the short period of life that intervenes between the appearance of recurrence and the death of the patient ; he finds this to average about 12 months for all stages of growth, and that in cases in which recurrence has been delayed

five or 10 years the prognosis is just as grave. On the other hand, Bryant's patients quoted above where recurrence took place 25, 31 and 32 years respectively, survived more than five years, and I have had cases of my own who have survived recurrence even as long as 10 years. The *"immunity" which had broken down seemed to have been re-established* in these more fortunate cases.

The vagaries and variations of this cancer immunity or resistance are to be occasionally seen in the recurring alternation of efflorescence and retrogression or disappearance of cutaneous nodules and even of lymphatic glands. Preternaturally slow growth of a mammary tumour and the prolonged absence of metastases may be indicative of the same cancer resistance ; the most remarkable example is that of Crivelli and Tinca,[20] whose patient had a carcinoma of the breast for 47 years, and only in the last two years of her life did the disease obtain the mastery. Still more amazing are the cases of *spontaneous disappearance of a cancerous mammary growth and its secondaries.* Sir Alfred Pearce Gould in his Bradshaw Lecture quoted a number of his own cases in which this phenomenon occurred, even when the patient had been almost *in articulo mortis.* Not a few of the cases mentioned by my old "chief" were personally known to myself, but I doubt not that many of the older surgeons here have been privileged to see such miracles. " Cancer, even when advanced in degree and of long duration, may get better, and does sometimes get well. Nature unaided may sometimes effect a cure." These statements are based on fact : I have not the wit or wisdom to weave them into theories, but I remain fearful of any form of treatment or any contingency which may even temporarily undermine the patient's resisting power to deal with residual malignant cells that may have been left behind at operation. Some of my patients with an axilla packed with infected glands, in whom it seems impossible that every cancer cell can have been removed, have remained well for periods of 15 to 20 years, in the absence of any postoperative X-Ray therapy.

Forty years have passed since I performed my first radical mastectomy, and although I have been attached synchronously to a plethora of hospitals, I confess to my surprise in June, 1938, to find that I had performed 603 radical operations for carcinoma mammæ. The year that intervened between the Plymouth meeting of the British Medical Association in 1938 and the outbreak of hostilities added another 25, but the war years where my function was that of a consultant rather than an operating surgeon, and those of more recent wanderings have only yielded an additional 17 radical ablations. My operative experience[21] therefore amounts to 645 radical operations for mammary cancer, while less than 100 patients' cases have, for various reasons, been treated by radiation only, a very few by limited operation and radiation or dismissed without active therapy. The figures which follow are the story of one surgeon's experience, and the class of patient that has come under my notice has, on the whole, been favourable.

*Total Personal Cases of Primary Carcinoma of the Breast, from
December,* 1907 *to December,* 1947, treated by Radical Operation,
Radiation alone, removal of the Breast and Superadded Irradiation
or no therapy : under   750

*Total Cases of Primary Cancer of the Breast dealt with by Radical
Operation from December,* 1907 *to December,* 1947   645

*Total Cases of Primary Carcinoma of Mamma submitted to
Radical Mastectomy up to June,* 1938   603

Cases of Radical Mastectomy from June, 1938 *to September 3rd,*
1939   25

Cases from September, 1939 *to December,* 1947   17

Total   645

Lazarus-Barlow and Campiche[22] in a statistical survey of 1,976 cases of
mammary cancer from The Middlesex Hospital records up to 1904
estimated that the average *natural duration of the untreated disease* was
25·4 months in cases with a large amount of fibrous tissue and few cells
and $5\frac{1}{2}$ years in the more cellular growths.   *The average natural duration*
of all cases was computed to be $3\frac{1}{2}$ years—a figure with which Rowntree[23]
is in agreement. It would, therefore, be almost impertinent in this assembly
of surgeons to discuss five-year results, and I will therefore confine my
statistics to 10-year survival cases.   It is refreshing to find Truscott in
his recent paper also considering the fate of patients 10 years after
treatment.

Total Cases of Radical Mastectomy up to 1928, i.e., cases
that could have survived 10 years or more   363

Group I	Cases operated up to 1928	113
	Cases surviving 10 years	95
	Percentage surviving 10 years	84·07
Group II	Cases of Radical Mastectomy up to 1928 ..	204
	Cases surviving 10 years	60
	Percentage surviving 10 years	29·4
Group III	Cases operated up to 1928	46
	Cases surviving 10 years	3
	Percentage surviving 10 years	6·5

By August, 1939, 169 of all my 388 patients (43 per cent.) in whom
radical mastectomy had been performed up to 1929, had survived 10 years,
many being of course still alive at that time.

Survival between 25 and 30 years   8
Survival of 20 to 25 years   6
Survival of 15 to 20 years   33
Survival of 10 to 15 years   122
169

Where information is lacking as to the histological grading of the breast tumour, no two series of cases or of treatments can be fairly compared. Far be it from me to vaunt the results of my own obstinate old-fashioned surgery or challenge my findings against those of other men and of other methods ; almost without exception the conclusions of the surgeon-radiotherapist " combines " are based on a five-year survival basis, which is not a very ambitious yard-stick, when the length of the natural history of mammary cancer is borne in mind. When their figures at 10 years convincingly surpass those which I have given from radical surgery alone, I may be prepared to solicit the routine aid of radiotherapy for the Group I and Group II patients.

" Grow old along with me,
The best is yet to be,
The last of life, for which the first was made."

One of the compensations of advancing years is the opportunity afforded of seeing patients again and again, especially those operated on for malignant disease several decades before. Sir James Barrie once said that " God gave us memory that we might have roses in December." To-night I would venture to say that the 169 patients who survived a radical mastectomy for cancer 10 years are " my roses in December."

Those who have served in the Royal Navy doubtless need no reminder of Drake's Prayer. " O Lord God, when thou givest to thy servants to endeavour any great matter, grant us also to know that it is not the Beginning but the Continuing of the same until it be thoroughly finished, which yieldeth the True Glory. Amen." This might well be placed on the walls of every Follow-Up Department. The ever-bloody Boche in April, 1941, destroyed my case-records and all my follow-up material, but the Prayer above quoted should act as a stimulus to me in my " sere and yellow " to endeavour to ascertain the subsequent history of those of my 169 patients who had survived 10 years after radical mastectomy *and who were still alive in* 1939. Glimmerings of the story of a few have stolen up from the distant past. To-day one lady with several infected glands in the axilla at the time of operation is alive 37 years after : another was run over and killed in a Gloucestershire street accident 36 years after operation ; a third died 36 years after the initial operation from what may have been an abdominal recurrence 36 years after ; while a fourth is alive and well just over 33 years after removal of a carcinomatous breast.

Considerations of time preclude me from dealing with the details of operative technique, or paying adequate tribute to George Grey Turner in this, as in every branch of surgery; there are no minutes left to discuss the treatment of pain, the œdematous arm, the occasionally dramatic effects of stilbœstrol, the value of sterilization by radiotherapy or the baleful results of pregnancy during the development of the tumour and the relation of any subsequent cyesis to recurrence, etc. The patient with enlarged supraclavicular glands will live longer if treated by radiation therapy than by any operative attack. The most profound anatomist and

skilful surgeon cannot perform a " block-dissection " of the supraclavicular fossa.

The hopes and aspirations of each one in this hall re-echo those lines in Wordsworth's ode " Intimations of Immortality."

> The Youth, who daily further from the East
> Must travel, still is Nature's Priest,
> And by the vision splendid
> Is on his way attended;

" the vision splendid," which for us is that long-desired day, perhaps not too far distant, when in the cure of cancer gross mechanical destruction and cruel mutilation of human tissues shall no longer be required, nor the scorching methods and machinery of Hephaestus the blacksmith god, or Prometheus who stole fire from heaven! "When the biologist shall know the laws that govern cell-growth with a knowledge akin in its sweep and accuracy to that of the astronomer," he will then have a power denied to those who scan the stars in the firmament, and that power will enable him to prevent, to control, and to cure cancer. *Lentement, mais toujours, l'humanité réalise les rêves des sages.*

I have spoken long enough

> " For I have neither wit nor words, nor worth,
> Action, nor utterance, nor the power of speech,
> To stir men's blood: I only speak right on.

In the long and toilsome effort along diverse paths to gain the shining peaks of victory, I give you all, my friends, radiotherapists, biochemists, my surgical brothers, I give you the ancient salutation: " Be of good cheer !"

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CONGENITAL ATRESIA OF THE ŒSOPHAGUS

Hunterian Lecture delivered at the Royal College of Surgeons of England

on

16th January, 1947

by

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INTRODUCTION: PATHOLOGICAL TYPES

SO MANY VARIETIES of congenital malformation of the œsophagus have been described that the true nature of the least rare variety of all is often overlooked; this is an atresia or interruption in the continuity of the œsophagus, with, usually, a fistula between the trachea and the distal segment. In 1929 Vogt made a classification of the different types of atresia:—

TYPE   I.—Complete absence of œsophagus.

TYPE II.—Atresia of the œsophagus with an upper and lower œsophageal segment, each ending in a blind pouch.

TYPE III.—Atresia of the œsophagus with tracheo-œsophageal fistula.

    (A) With fistula between upper segment and trachea.

    (B) With fistula between lower segment and trachea.

    (C) With fistula between both segments and trachea.

Eighty per cent. of all cases of atresia are of Type III(B), Fig. 1. That is to say, the upper segment ends blindly at about the level of the vena azygos arch, or 10 to 12 cm. from the anterior alveolar margin; it is dilated and hypertrophied and remarkably constant in size and shape. The lower segment shows more variation; it springs from the back of the trachea, usually within 1 cm. of the bifurcation, or from the bifurcation itself; within these limits it varies considerably, so that it may overlap the upper segment or it may be separated from it.

The lower segment is nearly always thin-walled and usually about half the calibre of the upper segment. It may be of nearly uniform diameter throughout its length or it may taper as it approaches the trachea.

These characteristics and variations are of great importance in determining the feasability of anastomosis.
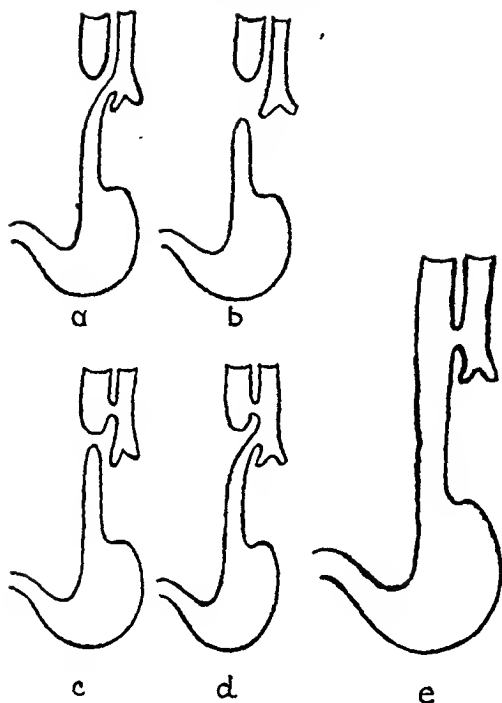
Fig. 1.

(a) Type III(B), the most common type, accounting for
80 per cent. of all cases.
(b) Next most common type.
(c), (d) and (e) All rare.

Type II accounts for most of the cases which do not fall into the characteristic group. Type III(A) and Type III(C) are rare.

Cameron Haight encountered an example of a single fistula between œsophagus and trachea which was not associated with atresia.

Historical Note

Gibson, Physician-General to the Army and a grandson of Oliver Cromwell, described this condition in 1697 and it was mentioned by Durston some 27 years earlier.

About 250 years elapsed before successful methods of management were devised and carried out. The subject was not forgotten during this time. Martin published a case report in 1821, and in 1884 MacKenzie was able to collect records of 43 cases. In 1910 Sir Arthur Keith searched the museums of London and found 14 examples of atresia. He described accurately what we now know to be the most usual type, and gave a satisfactory account of the developmental errors underlying the condition.

The only type of operation carried out at this period was a simple gastrostomy.

Honourable exception must be accorded to Richter, who attempted a rational operation on two infants in 1913, a year memorable for Torek's successful removal of the thoracic œsophagus for carcinoma.

By 1933 some 300 cases had been reported in the literature; most of the reports emphasised the rarity and hopelessness of the condition, while a few of the more adventurous expressed the pious hope that the condition might one day yield to surgery.

Two popular misconceptions have discouraged attempts at treatment, first the alleged extreme rarity of the lesion, and second the mistaken idea that this anomaly is usually associated with other and equally serious malformations.

The second misconception is the more insidious in that it gives that final stamp of hopelessness to what is already a very difficult surgical problem. It is true that Plass (1919) found that about half the recorded cases show associated anomalies. But it must also be remembered that in 45 consecutive cases, Cameron Haight found that only one patient had an additional deformity which was incompatible with life.

The possible presence of other abnormalities, particularly imperforate anus, must be recognised, but this recognition must not be used as an argument for regarding the condition as hopeless.

Most authors have commented on the extreme rarity of the condition —the popular figure given being 1 in 50,000 births. Guthrie (1945), searching the material at the Royal Hospital for Sick Children, Glasgow, found that between 1915 and 1944, in 6,916 consecutive post mortem examinations, malformations of the œsophagus were found in 38 subjects, and no fewer than 24 of these provided examples of atresia (1 in 284 autopsies). Six other patients who did not come to autopsy presented suggestive clinical features. At the Postgraduate Medical School of London, from 1935 to 1942, four cases of atresia were found in 10,543 deliveries, or one in 2,635 deliveries. When it is considered that nearly every one of the autopsy cases investigated by Guthrie showed pulmonary complications of great severity, it is reasonable to assume that a number of these infants succumb without the true nature of the lesion being recognised.

The Surgical Problem

Attempts at feeding fill the blind upper segment so that its contents spill over into the trachea to evoke attacks of cyanosis, and, before long, aspiration pneumonia. Even if feeding is avoided, the blind upper sac fills with mucus and saliva unless aspiration is carried out at frequent intervals. Gastrostomy feeds too find their way into the lungs through the distal œsophagus and its tracheal fistula, unless the anomaly is one of the rare forms in which there is a blind lower segment. Apart from the fact that gastric contents may pass into the lungs with fatal results, air passing from the trachea into the stomach sometimes causes considerable gastric

distension. A type of spurious or see-saw respiration may even be observed, air passing backwards and forwards from the lungs to the stomach by way of trachea, fistula and œsophagus ; this gastric respiration produces a rapidly increasing anoxia.

In an attempt to save these infants, Steele carried out gastrostomy in 1888. Others followed his example, but all gastrostomies were unsuccessful because of the regurgitation into the lungs. Brenneman (1918) hoped to overcome this difficulty by substituting a jejunostomy for gastrostomy, but again without success. Richter, in 1913, recognised the importance of the fistula and advocated transpleural ligature of the lower œsophagus
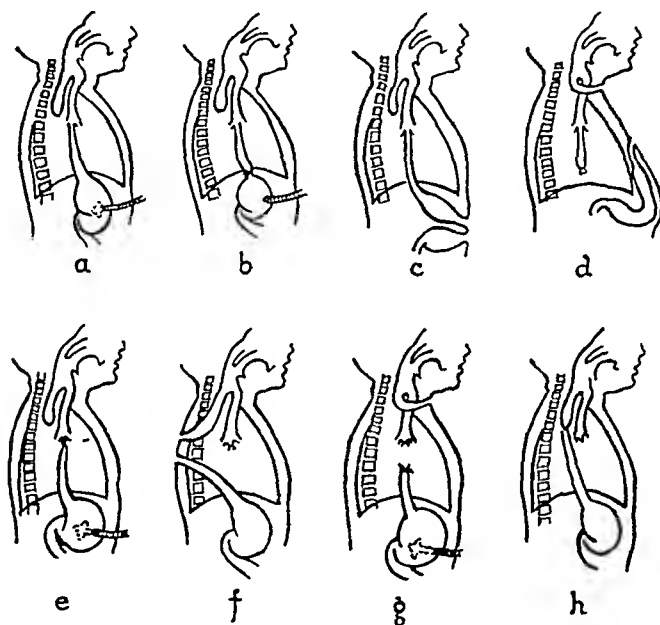
after Humphreys.

Fig. 2.

Stages in the development of the operation.

(*a*) Simple gastrostomy.

(*b*) Gastrostomy + ligature at cardiac end of the œsophagus.

(*c*) Formation of a gastric spur.

(*d*) Formation of stomach tube together with ligature of lower œsophagus.

(*e*) Gastrostomy + direct approach to the fistula and ligature.

(*f*) Exteriorization of both segments.

(*g*) Gastrostomy + œsophagostomy and ligature of fistula.

(*h*) Present operation—direct approach and anastomosis.

together with gastrostomy. His two cases were unsuccessful, and it must be noted that he made no provision for keeping the blind upper segment empty. Direct anastomosis by means of the Murphy button was advocated by Von Hacker in 1926 (quoted by Lanman, 1940), but without success.

By 1929 there was more general realisation of the nature of the problem, and its solution was seen to require :—

I. Prevention of the blind upper segment from filling up and overflowing.

II. Prevention of the regurgitation of gastric contents through the fistula into the trachea.

III. Successful feeding of the infants.

During the next few years all sorts of methods were adopted to achieve these results, and the names of Mixter, Leven, Gage and Ochsner, Gamble and Ladd are all associated with multiple operations which include cervical œsophagostomy, various types of gastrostomy, transection of the stomach and ligature of the fistula. Out of all these attempts only two were completely successful at this time ; the staged operations performed by Ladd in 1939, and Leven in 1940.

The multiple-stage operation presents the patient and the surgeon with so difficult a succession of hurdles even if the infant survives the early procedures, that Lanman ventured a primary anastomosis by the extrapleural route in 1936, but none of his patients survived.

Haight and Townsley reported in 1941 the first completely successful direct anastomosis, and the tendency now is to favour direct anastomosis wherever possible. The development of the operation is shown in Fig. 2.

The transpleural and extrapleural routes have each had their supporters. Haight and Townsley used the latter in their first successful case. The anastomosis is less difficult by the transpleural route and the operation seems to be well tolerated, but up to the present time this approach has, generally speaking, proved unsuccessful, although Singleton and Knight report the case of a patient who lived for over seven months following the transpleural operation, and whose death then was probably due to a fistula of the trachea, unsuspected during life, with the upper blind segment of the œsophagus (Vogt's Type III(c)).

Impressed with the difficulties which I experienced in carrying out the extrapleural operation, both in the post mortem room and in the operating theatre, I concentrated at first on the right transpleural approach, but have been forced to the conclusion that in the anastomosis of such a delicate structure as the infant's œsophagus, it is difficult to avoid leakage, and if the leak occurs inside the pleural cavity it is likely to be fatal. My

own experience, which has impressed me with the value of the extra-pleural procedure, has been gained in the following cases :—

OPERATIONS ON CASES OF CONGENITAL ATRESIA OF OESOPHAGUS
1938-1946. FRANKLIN

Case	Date	Sex and Age (Hours)	Birth Weight and Type	Procedure	Survival	Remarks
1	21.11.38	M. 72	6lb. 7oz. III(B)	Ligature of cardia and gastrostomy	7 days	Silk ligature slowly cut through and re-established the fistula
2	16.10.41	M. Prem. 84	4lb. 5oz. III(B)	Transpleural (R). Anastomosis	17 hours	Pneumonia
3	31.5.42	F. Prem. 148	5lb. 8oz. III(B)	Transpleural (R). Fistula ligated and divided, tube tied into lower segment	Died on table	Massive collapse of both lungs
4	2.11.43	F. Prem. 72	4lb. 3oz. III(B)	Transpleural (R). Anastomosis	5¼ days	Leakage of anastomosis. Case reported (O'REILLY, N., FRANKLIN, R. H., and DALEY, M. D., 1944)
5	6.11.45	F. Prem.	III(B)	Transpleural (R). Tube tied into each segment	43 hours	Pneumonia
6	13.1.46	F. 28	5lb. 8oz. III(B)	Transpleural (R). Fistula ligated and divided. Gastrostomy	52 hours	Pneumonia
7	21.2.46	F. 120	5lb. 6oz. III(B)	Extrapleural approach attempted. Pleura tore badly. Anastomosis with difficulty	27 hours	Pulmonary collapse
8	27.10.46	F. 72	7lb. 2oz. II	Transpleural (R). No fistula. Lower segment ended 1·5 cm. above diaphragm. Gastrostomy	25 hours	Consolidation both lungs. Patent ductus arteriosus. No gastric gas bubble was seen at X-ray—this should have led to the correct diagnosis of type
9	10.1.47	F. 72	7lb. 7oz. III(B)	Extrapleural (R). Anastomosis	Alive and well	Gastrostomy carried out 6 days after anastomosis. Gastrostomy tube removed after 12 days. Infant now takes food normally and has made good progress. (Previously reported, FRANKLIN, R. H., 1947)
10	24.4.47	F. 48	6lb. 7oz. III(B)	Extrapleural (R). Anastomosis	Alive and well	Gastrostomy was not necessary in this case. The infant made a smooth recovery and has since progressed well. (Previously reported, FRANKLIN, R. H., 1947)

Diagnosis

Cyanosis associated with excess of mucus in the nasopharynx may be noted at birth in many normal infants. Improvement in these occurs after aspiration, but the child suffering from atresia has recurrent attacks of cyanosis which are usually severe enough to excite notice, but which are often attributed to other causes, such as tentorial tears and atelectasis. When feeds are started all the symptoms are accentuated, *the child is* often avid for its feed and sucks strongly, only to be overwhelmed by an attack of spluttering and cyanosis, accompanied by the complete regurgitation of the feed. Aspiration of the nasopharynx effects an immediate improvement, but the symptoms recur as soon as feeding is attempted again.

These symptoms are so striking and alarming that they seldom escape notice, and it cannot be too strongly emphasised that such a train of symptoms usually indicate œsophageal atresia. Midwives and others who have the care of infants in the first hours of life, should be taught that atresia is the commonest cause of persistent choking and cyanosis in the newborn child.

Once the suspicion of atresia has been raised, all mouth feeding is stopped and the nasopharynx is aspirated at frequent intervals.

To confirm the diagnosis, a well-lubricated rubber catheter is passed through the mouth and down the œsophagus. Its arrest 10-12 cm. from the anterior alveolar margin is practically diagnostic of atresia.

Final confirmation of the diagnosis is radiological, but the practice of giving a Barium swallow is to be condemned. The emulsion is almost certain to spill over into the lungs and the child's slender chance of survival is still further reduced. The best method to adopt is as follows :—

The infant is screened and a careful assessment is made of the condition of the lungs ; the stomach and intestines are then examined for the presence of air. If air is present in the stomach and intestines below an œsophageal atresia it is proof that a fistula exists between the lower segment and trachea. The converse is usually, but not invariably, true.

A catheter is now passed into the upper segment and 1 to 2 cc. of lipiodol is gently introduced under direct observation against the screen.

The appearance of the blind segment is characteristic, and as soon as it has been observed and the rare presence of a fistula in the upper segment excluded, the lipiodol is withdrawn (Figs. 3, 4 and 5). Bronchoscopic examination has been used to inspect the fistula and so confirm the diagnosis (Pilcher). Apart from the passage of a catheter into the blind sac and frequent aspiration, the important features in the management of the diagnostic stage are negative rather than positive. The chief safety factors are the avoidance of any further feeding and of unskilled attempts at X-ray diagnosis.

Management after the Diagnosis has been Confirmed

The complete diagnosis not only confirms the presence of atresia, but in many cases discloses the precise nature of the deformity. It must be emphasised once more that 80 per cent. of the cases fall into the characteristic group of œsophageal atresia with fistula between trachea and lower œsophageal segment.

The preparation of the patient for operation requires the full collaboration of an expert pædiatrician, as well as the whole-time services of an intelligent and conscientious nurse. In fact, without these two essentials before and after operation, any surgical intervention is almost certain to fail.

The three essentials in the pre-operative period are : to keep the blind sac empty by aspiration, to improve the state of the lungs by changing the child's position frequently and systematically, and to administer the

Fig. 3

X-ray showing large air bubble in stomach and blind upper œsophageal segment filled with iodised oil.

Fig. 4

Blind upper segment filled with iodised oil. Care has been taken not to allow any to spill over into the larynx.


Fig. 5

An earlier case in which the importance of great care in X-ray diagnosis was not fully appreciated and the iodised oil has spilled over into the lungs. Although the œsophagus was restored and is seen to be functioning, the pulmonary condition contributed to the unsuccessful result in this case.

requisite fluid parenterally. The fluid requirement is difficult to assess, and giving too much is probably more dangerous than giving too little.

The operation

The child is secured in a prone position over a rubber hot-water bottle, half-filled with warm water. A small pad is placed under the right shoulder and the head is turned to the right. Local anæsthesia (1 per cent. Procain) is used for the skin incision and the anæsthetist concentrates on keeping the upper œsophageal segment empty and administering oxygen under pressure from time to time.

The incision is curved (Fig. 6), starting 1 cm. lateral to the spine, over the second rib, passing downwards parallel to the spine over the third and fourth ribs and then outwards crossing the fifth rib obliquely and ending over the sixth rib. Part of the fifth rib is resected subperiosteally, extreme care being necessary to avoid opening the pleura. The correct extrapleural plane having been found, the incision is deepened through the chest wall with division of the fourth, third and second ribs and their intercostal bundles. The pleura is carefully displaced until the vena azygos arch is seen. This usually marks the lower end of the upper blind segment and it is divided between ligatures with great care.
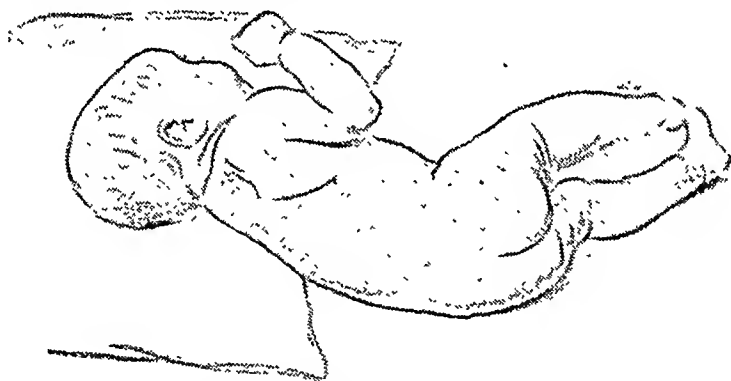
Scar after mediastinal incision.

Fig. 6

The lower œsophageal segment emerges from the posterior surface of the trachea or from the bifurcation itself. Gentle movement of the catheter in the upper segment helps in identification. The fistula is ligatured close to the trachea and divided, with conservation of as much as possible of the lower segment, and traction sutures are introduced into each segment to avoid handling them unnecessarily.

Anastomosis is undertaken if it seems feasible without undue tension, and it is performed with the use of fine silk and small, curved, eyeless round-bodied needles. Various methods of anastomosis have been described, but none of them is easy. The structures are delicate and

readily damaged, and there is a great tendency for the stitches to cut out. A catheter introduced into the œsophagus from the mouth during the later stages is of great help. On completion of the anastomosis the area is well dusted with penicillin powder, the wound is closed around a small rubber drain, and the œsophageal tube is removed.

If anastomosis has proved impossible, Humphreys has advised that the first stage of a multiple-stage operation be carried out. A finger is passed along the back of the trachea into the base of the neck near the suprasternal notch. An incision is made between the right sternomastoid and the trachea, and through this the upper segment is drawn to establish a cervical œsophagostomy. If a fistula is present it is ligated and divided at this first stage, but failure to effect an anastomosis may be due to the absence of a fistula ; the blind ends of the œsóphagus being further apart when atresia is uncomplicated (Type II) than when a tracheo-œsophageal fistula is present as well (Type III(B)).

After-care

The child is nursed in an oxygen tent and the same care is taken to aspirate the nasopharynx as before operation. A small blood transfusion (100 cc.) may be given, followed by an intravenous drip of N/5 glucose saline. The fluid and salt balance must be controlled with great care and is best entrusted to a skilled pædiatrician.

Immediately after the operation, feeds of penicillin solution (10,000 units) hourly are started by mouth. The mediastinal drainage tube is removed on the third or fourth day and the patency of the œsophagus is tested radiographically with iodised oil ; if the reconstructed œsophagus conducts the oil without leakage, feeds of expressed breast milk are begun.

Gastrostomy is performed only if the swallow of iodised oil demonstrates a mediastinal leak ; even if it is shown to be necessary by this criterion, its performance is best deferred until the seventh or eighth day after œsophageal repair.

Results in Other Centres

Haight's results to 1946 :—

> 45 consecutive patients.
> 42 had associated tracheo-œsophageal fistula ; one additional patient had a tracheo-œsophageal fistula without œsophageal atresia.
> 36 patients were explored.
> 26 patients were submitted to intrathoracic restoration of œsophagus.
> 9 patients survived operation from 6 months to $4\frac{3}{4}$ years.
> > Only one patient in this series had an additional anomaly which was incompatible with life.

Ladd's results to 1944 :—

34 patients explored.

28 patients submitted to a staged operation ; nine living.

6 patients treated by primary anastomosis ; two living.

Ladd's first success was a staged operation in November, 1939 ; an antethoracic œsophagus was constructed and the child was reported alive at the age of 4¼ years.

Conclusions

This paper is presented in the hope that midwives and pædiatricians will regard all newborn infants who suffer from attacks of cyanosis and choking, together with the regurgitation of feeds, to be subjects of atresia of the œsophagus until the œsophagus can be shown, by the clinical and radiological methods described, to be normal.

If this attitude is inculcated and lethal attempts at feeding are stopped until the condition of the œsophagus has been determined by the simple passage of a catheter, we shall have more frequent opportunities of treating these little patients before aspiration pneumonia has jeopardised their chances of recovery.

The radiologist can contribute to the success of the operation by refraining from any form of examination which risks filling the lungs with opaque medium and the surgeon can prepare himself for a difficult operative procedure by a study of the varied pathology of œsophageal atresia and of the methods which have proved successful in its treatment.

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TUMOURS OF STRIATED MUSCLE

Post-graduate lecture delivered at the Royal College of Surgeons of England,
on
9th September, 1947
by
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IN SPEAKING of rhabdomyoma I propose to review the whole group of tumours, simple and malignant, which are thought to be composed wholly of striated muscle cells or of elements derived from them. I do not propose to include the complex tumours in which striated muscle is one of a number of neoplastic tissue elements, *e.g.*, in the mixed tumours of kidney and testis and the still more complex teratomata. Since my audience is interested chiefly in surgery I shall devote most of the time to conditions of surgical importance.

At the outset we are faced with the difficulty of terminology, for certain neoplasms arising in striated muscle may present the morphological and tinctorial qualities of muscle substance but lack unequivocal cross-striation. Such tumours are appropriately classified as *Myoblastoma* (Cappell and Montgomery, 1937) and this type is commoner than is generally realised.

The term myoblastoma has unfortunately been used in different senses and there is often difficulty in distinguishing precisely which class of tumour is under discussion (*e.g.*, Howe and Warren, 1944).

It would be an advantage if all tumours which show unequivocal, even if imperfect, cross-striations were classified as rhabdomyoma, and it is unfortunate that the majority would merit the qualification " sarcomatodes." Abrikossoff's type four (polymorphous myoblastic sarcoma) should certainly be put in this group. On the other hand his type one or pure myoblastoma, composed of cells with a strikingly granular cytoplasm devoid of striations but tinctorially resembling muscle, is sufficiently different in histological character and clinical behaviour to merit separate consideration and I shall distinguish them as the " granular-cell myoblastoma."

Rhabdomyomata of apparently simple character occur in the heart muscle, in association with other congenital malformations, especially with tuberous sclerosis of the brain. In all other sites striated muscle tumours are usually malignant, giving rise to repeated local recurrences followed by metastases to the regional lymph nodes and finally by generalised blood-borne dissemination. The distinction between rhabdomyoma and myoblastoma is purely histological and is very ill-defined, since cross-striations are often imperfectly developed and require special technical methods for their detection ; further they may be found in only a single part of the growth or only in a metastasis. I shall therefore consider them together regionally and illustrate them from my own cases.

Striated muscle tumours have been reported in a wide variety of sites but the commoner locations are the skeletal muscles, beneath the mucosa of the oropharynx, and the urogenital tract. Table I shows the distribution of cases observed personally.

TABLE I

Rhabdomyoma		Myoblastoma	
Palate region	5	Pharynx and larynx	2
Diaphragm	1	Orbit	2
Deltoid muscle	1	Bladder	2
Cervix uteri	1	Spermatic cord	2
Prostate	3	Pleura and chest wall	2
		Miscell. Muscles	10

Tumours of Skeletal Muscles

The general features of this group have been described and beautifully illustrated by Gordon-Taylor (1940) and by De and Tribedi (1940), and a table of their topographical distribution compiled by Stout (1946) has been summarised in Table II, with the addition of my own new cases. A considerable proportion of the tumours arising within the substance of skeletal muscles are myogenic and can be distinguished by their histological characters, and I do not think they are so rare as Gordon-Taylor suggests.

TABLE II

Tumours of Skeletal Muscle.

Lower limb	Groin	4	
	Thigh	38	
	Gluteal	7	67
	Leg and foot	18	
Upper limb	Axilla	3	
	Arm	18	26
	Forearm	5	
Head and neck	Face	3	
	Neck	6	
	Tongue	10	25
	Palate	6	
Trunk	Chest	5	
	Back	11	
	Diaphragm -	4	23
	Abdom. Wall	2	
	Psoas	1	

The naked-eye appearances are not highly characteristic and vary considerably from case to case. In the main these tumours are of typically sarcomatous aspect, in varying shades of pink and cream colour with

areas of necrosis, hæmorrhage and cystic degeneration. De and Tribedi record several cases as of hard, almost cartilaginous, consistence but I have not observed this and am more impressed with the occurrence of soft myxoma-like tissue. In general the growths form bulky swellings, oval or elongated within individual muscles at first and unfortunately they may then look as if encapsulated, but this is a deceptively false appearance. Characteristically they are at first mobile when the muscle is at rest but become fixed on contraction. Later they infiltrate adjacent muscles and connective tissue planes and become adherent to surrounding structures even eroding bone and simulating osteogenic sarcoma. Invasion of the skin is followed by fungation especially after biopsy. Excision is very frequently followed by local recurrence and even amputation may not suffice, for recurrence in the stump has been observed, indicating local spread far beyond the limits of naked-eye recognition. In this the malignant rhabdomyomata differ from most other sarcomata in that they spread by lymphatics to regional lymph nodes, and ultimately, dissemination by the blood stream leads to metastases in the lungs and internal organs and especially in the vertebræ (Cappell and Montgomery, 1937 ; Veits and Wittenberg, 1945). There is, however, characteristically a considerable period, often extending over years, during which repeated local recurrences take place and there is therefore the strongest incentive to thorough and radical treatment at the earliest moment. The pseudo-encapsulation of the early stages is tempting, but as Gordon-Taylor points out enucleation has no place in the treatment of these tumours and timid or inadequate excision is likely to be disastrous. Here, as in the neuro-fibroma group, 1 am profoundly impressed with the value of a bold sacrifice at the first opportunity as giving a greater hope of freedom from recurrence. Removal of the whole of the affected muscle group and an ample margin of surrounding tissue gives the only hope short of amputation.

Histologically these neoplasms are composed of very pleomorphic cells :—

(a) Spindle-shaped elements and tubular or strap-like cells showing longitudinal and transverse striation in greater or lesser degree ; these commonly show several nuclei arranged in a compact row, or two nuclei tandem position (fig. 7) ; the long, ribbon-like cells very commonly show acute angulation, a feature which I regard as of diagnostic significance.

(b) Racquet-shaped cells with a large nucleus in the rounded extremity and a long tapering process in which striations may be detected.

(c) Rounded cells with rather scanty cytoplasm and a single, large nucleus—highly anaplastic elements (figs. 4 and 5).

(d) Giant cells often bizarre in shape and with many nuclei ; often with highly vacuolated cytoplasm and sometimes with abundant hyaline acidophil droplets (fig. 13).

(a)   (b)   (c)

Fig. 1. (a) original tumour ; (b) first recurrence ; (c) third recurrence. $\times \frac{8}{9}$.

Fig. 2. Dissection of neck to show numerous hæmorrhagic and necrotic secondary growths in glands of left side. $\times \frac{2}{5}$.

Fig. 3. Lung on section, showing multiple secondary growths in parenchyma and in hilum glands. $\times \frac{3}{10}$.
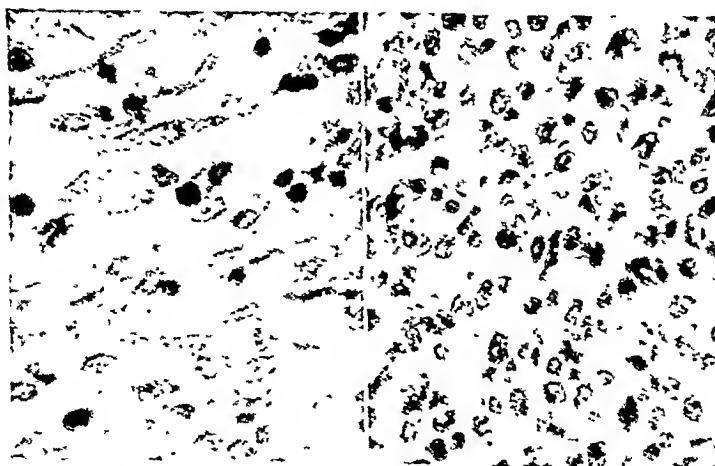
Fig. 4. Original tumour : (a) spindle cells and less differentiated myoblasts devoid of cross striation ; (b) " round-cell sarcoma " composed of very primitive, quite undifferentiated myoblasts. Masson's trichromic stain. $\times 390$
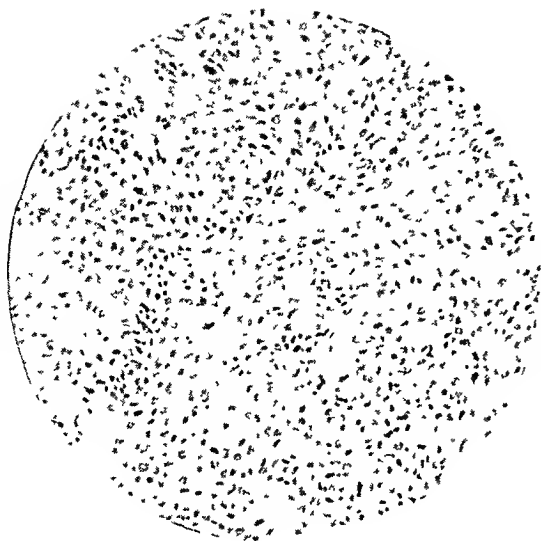
Fig. 5

Closely packed round cells with scanty cytoplasm form a solid nodule amongst the looser tissue × 150.

Fig. 6

Pseudo-myxomatous tissue. The fibres are the very long, fine processes of primitive muscle cells and the intercellular matrix does not stain like mucin. × 150.
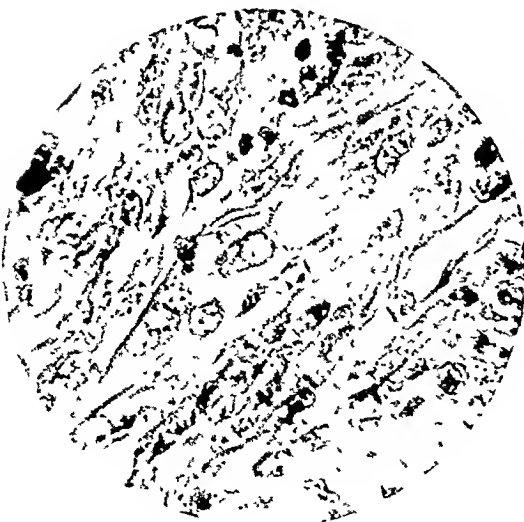
Fig. 7

Large myoblasts, some multi-nucleated; these show only longitudinal striation.
× 400.

Fig. 8

Lymph-node metastasis. The round cells are arranged in an alveolar architecture, emphasised by silver impregnation of the reticulum. Foot's method (modified).
× 185.
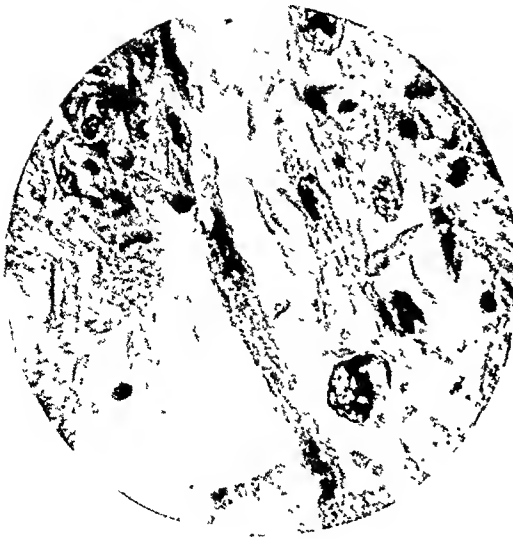
Fig. 9. Case 3—A strap-like cell with well marked longitudinal striation crosses the field obliquely. Above it are granular cells, below it a spindle cell with forked extremity is seen. Celestin blue azo-eosin. ×390.

GRANULAR-CELL MYOBLASTOMA OF TONGUE

Fig. 10. The growth is composed of elongated syncytial masses of granular acidophilic protoplasm. The remains of a striated muscle fibre are seen, but transitions from normal to granular cells are rare. ×475.
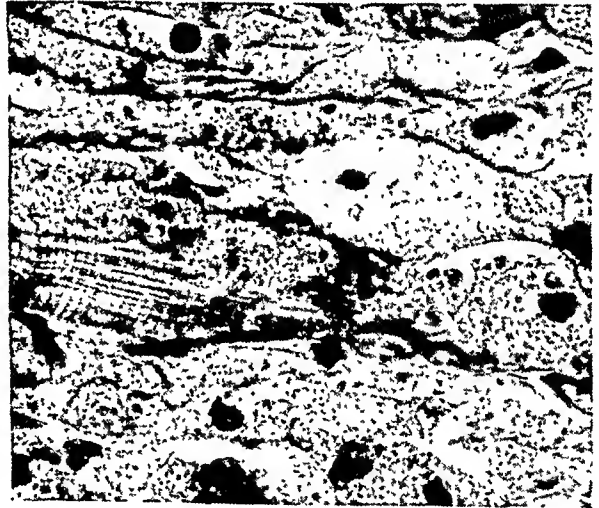
Fig. 11. There is irregular hyperplasia of the covering squamous epithelium with numerous epithelial downgrowths and cell nests. Note the resemblance to early squamous epithelioma. ×60.
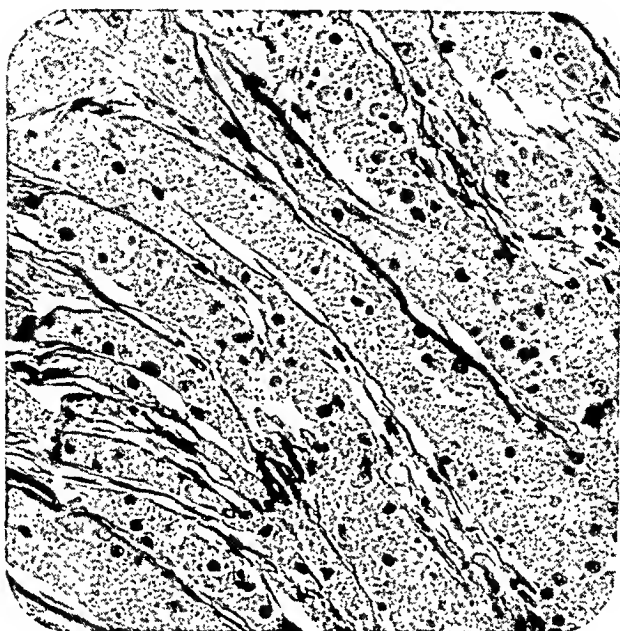
Fig. 12

The growth is composed of syncytial masses of highly granular acidophilic protoplasm with small darkly staining nuclei. ×110.

MYOBLASTOMA OF THE CHEST WALL
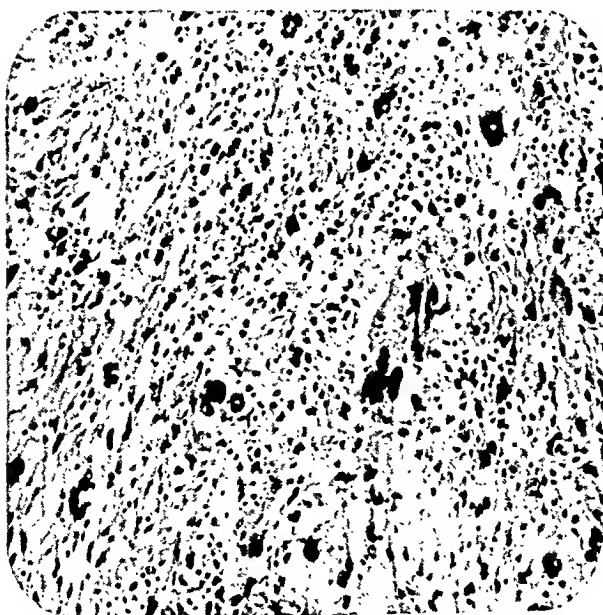
Fig. 13

The growth is composed of irregular spindle and strap-like cells, some of enormous size with huge hyper-chromatic nuclei. This type of " mixed-cell sarcoma " is often of muscle origin. ×110.

The nuclei of the cells often show a single large prominent nucleolus and are often rather poor in chromatin, and a conspicuous feature is the occurrence of rows of nuclei in the longer strap-like cells. The combination of fusiform cells and giant cells, the nuclei of which show coarse karyosomes and large nucleoli is very suggestive of an origin in skeletal muscle (Foot, 1945). Rakov (1937) insists that giant cells are an invariable constituent of the tumours and are of diagnostic importance; he states, however, that tumours predominately composed of round and spindle forms without giant cells are notably worse in prognosis, and with this opinion I am in agreement. As stated in 1937, I believe that highly anaplastic round and spindle cells are far more likely to be the source of dissemination than the giant myoblastic syncytial elements, but further differentiation may occur after implantation in a new site.

The abundant cytoplasm of the tumour cells is characteristically strongly acidophilic, staining brightly with eosin, vivid red in Mallory's acid fuchsin-aniline blue method and clear yellow in Van Gieson, yet in examples unequivocally proved by the demonstration of cross-striations I have observed that the acidophile state of the cytoplasm is variable and may be so much less striking in the more anaplastic portions as not *per se* to lead one to suspect the true diagnosis. To demonstrate striations Heidenhain's iron hæmatoxylin is excellent but difficult to control in the finest fibres, and Mallory's phosphotungstic acid hæmatoxylin is probably the most reliable method. The amount of stroma varies but a fairly characteristic feature is the presence of a fine reticulum sheath around practically every cell.

Growths of the skeletal muscles include some with well-defined longitudinal and cross-striation, but in the majority these features are infrequent and may be wholly absent. The tinctorial reaction of the cytoplasm and the morphology of the cells are then the decisive features in diagnosis but no absolute line can be drawn between rhabdomyoma and myoblastoma.

Two examples illustrate this group. Lendrum (1947) has recently reported a malignant rhabdomyoma of the diaphragm in a man with pulmonary asbestosis. A fleshy mass 7 cms. in diameter occupied the right cupola of the diaphragm and spread directly into the liver. There were widespread metastases in the pleura and in the abdomen and a notable feature was the failure to demonstrate cross-striations in the primary growth, whereas they were clearly seen in the secondaries in the pelvis.

The next case illustrates a growth in the commonest site, *viz.*, upper thigh. A man aet 39, observed a swelling in the antero-lateral aspect of the left thigh with increasing pain and tenderness during the last five weeks. A large ovoid swelling was present, exquisitely tender, semifluctuant and a little red and hot to the touch. The growth moved with the quadriceps. There was intermittent fever and marked loss of weight. At operation the growth was exceedingly soft and so vascular that only

fragments could be removed. The wound was packed to control bleeding and three weeks later a radical removal of the whole of the affected muscle group was undertaken. Healing was satisfactory and there has been no recurrence in two years.

Histologically this is a very bizarre tumour, consisting chiefly of enormous syncytial giant cells with multiple nuclei and abundant cytoplasm. Some are packed with rounded brightly acidophilic droplets, some show traces of longitudinal fibrillation but transverse striations have not been detected.

In the aetiology of such tumours trauma has of course been blamed, in most cases inconclusively. I have personally observed two orbital rhabdomyomas in young boys alleged to follow a blow on the eye. In the first case the growth was roughly dumb-bell shaped and about 3 cms. in length by 2 cms. in greatest diameter ; surgical removal was followed by prompt recurrence and fungation. Exenteration of the orbital contents failed to cure and death occurred within two years.

The second case, a lad aged 14, was struck on the eye at work ; some months later a mass appeared in the orbit and was removed surgically. In view of the rhabdomyomatous nature of the growth a poor prognosis was given, but there has been no recurrence over a period of seven years, and the boy has remained well to date.

Rhabdomyoma of the orbit has been recorded also by Calhoun and Rees (1942) and Holste (1942).

Striped Muscle Tumours below Mucous Membranes

In contrast to the lack of characteristic appearances of tumours in the skeletal muscles, rhabdomyomata developing beneath mucous surfaces present naked-eye appearances by which their nature may be suspected, *viz.*, their tendency to assume a polypoid form with numerous blunt clubbed processes. This is so constant a feature that the diagnosis can be made with a high degree of probability on clinical grounds. True rhabdomyomata occur beneath the mucosa of the palate and pharynx, the urinary bladder and of the cervix uteri, and all present this typical appearance.

Palatal Growths

I have seen five such growths in the palate and two in the pharynx, and examples from these illustrate the salient points.

Case I. W.S. f. aet 10. First seen in 1928 with a polypoid growth hanging by a narrow pedicle from the uvula. It was removed by snare. Recurrence did not take place until 1931, when the growth was again removed by snare. Nine months later speech was affected and a cauliflower-like recurrence covered the soft palate, left tonsil and left pillar of fauces. Removal by diathermy was followed by radium application and the wound healed. Recurrence again took place within a year despite intermittent exposure to radium and in December, 1933, removal of the

recurrent growth was undertaken by diathermy, the tonsil and posterior pillar of fauces also being removed. She remained well for over a year and no further local recurrence took place, but about 14 months after the last operation she died with generalised metastases in lymph nodes, lungs and bones.

Case II. In a boy of 13 years presents a similar naked-eye appearance and a similar clinical course of repeated recurrences terminating with cervical glandular and pulmonary metastases. The characteristic coarsely lobulated and clubbed processes were present and the growth contained both spindle-cells and fully striated elements.

These two cases are fully reported by Cappell and Montgomery, (1937).

Case III. A boy aet nine years presented a grossly thickened soft palate with " papillomatous " fringes along the free border. Complete surgical removal was impossible but biopsy revealed the nature of the growth and treatment by the radium beam was given. Marked local improvement was observed, but recurrence followed within four months and soon thereafter signs of metastatic growths in the lungs were detected. Death took place about one year from the onset of the symptoms.

Striped Muscle Tumours of the Urogenital System

The urogenital tract is one of the more common sites of tumours containing striped muscle, but I am not considering here the most frequent type, viz., that in which striated muscle is a constituent of a mixed tumour. I shall illustrate this group by reference to examples in the cervix uteri, spermatic cord, prostate gland and urinary bladder.

Rhabdomyoma of the cervix uteri is one of the best-known forms of this growth—the sarcoma botryoides of Pfannenstiel.

It occurs both in children and in adults ; in the former usually involving the vagina, whereas in the adult it affects the cervix. The tumour in the adult gives rise to a thickening of the cervix from which numerous polypoid growths extend into the vagina. Ulceration with bloody and fœtid discharge usually occurs and repeated local recurrences are the rule. Ultimately metastases to the lungs are likely to supervene. This group is illustrated by the following case, the history of which is fairly typical :—

J. H. f. aet. 55. Complained of irregular bleeding of four months duration. A bulky tumour of the cervix was removed and was thought to be leiomyosarcoma. Recurrence following within three months giving rise to a fungating lobulated polypoid growth springing from the posterior lip of the cervix on the right side. Further biopsy showed the growth to contain striped muscle cells. Radium treatment was given. 2400 mgm. hrs. to the uterus and 4700 mgm. hrs. to the vaginal vault. The growth regressed in the cervix and vault of the vagina, but within six months pelvic infiltration appeared and a large abdominal mass was palpable. Œdema of the lower limbs followed and death occurred about one year after the onset of symptoms.

Microscopically the tumour is extremely cellular and in most parts presents the appearance of a large round-cell sarcoma with hyperchromatic nuclei and scanty cytoplasm. In places, however, the cells assume a spindle form and in a few areas are tubular or ribbon-like with the characteristic acute angulation of the fibres. These cells show distinct longitudinal and transverse striation but throughout most of the growth such diagnostic features are absent.

Rhabdomyoma of the uterine body is much rarer, but it presents the typical morphology of clubbed polypoid processes protruding into the uterine cavity and distending it with consequent irregular bleeding. Cases are reported and the literature reviewed by Gunning and Ross (1940) and by Campbell (1940), the latter having collected 21 cases.

Myoblastoma of the Spermatic Cord

J. R. aet 52. After a trivial injury bilateral hydroceles appeared and on the right side there was a firm ovoid tumour of the spermatic cord about 6 × 5 cms. in greatest length. This was removed together with the testis and cord and an enlarged inguinal lymph node. Recovery was uneventful and there was no recurrence six years later.

W.B. aet 52. Developed a mass in the right spermatic cord which had recently become painful. Surgical removal was followed by radiation treatment and so far as is known the patient has remained well.

Both tumours consist of spindle-shaped and tubular cells with characteristic large vesicular nuclei sometimes arranged in tandem in the elongated cells. The staining reactions of the cytoplasm are characteristic of muscle, and longitudinal striation is present but transverse striation is not demonstrable.

Cases of fatal metastasising rhabdomyoma have recently been described in this region : in the spermatic cord (Shivers, 1944) ; in the epididymis (Strong, 1942), in the testis (Beard and Hewit, 1945) ; and Prince (1942), collected 15 testicular cases in the literature and added a new example.

Rhabdomyoma of the Prostate Gland

Striped muscle tumours of the prostate are encountered chiefly in children, and it is probable that a considerable proportion of the sarcomata of the prostate in children are of this nature. Shattock drew attention to the presence of striated muscle in the postero-superior part of the prostate in the fœtus and in children and the relative frequency of striped muscle growths of the prostate in early life suggests that intrinsic developmental factors are more important here than exogenous influences.

The characteristic clinical features of rhabdomyo-sarcoma of the prostate are signs of bladder neck obstruction, followed by forward and upward displacement of the bladder and trigone. Fullness in the rectum is an early sign, with a rather soft elastic tense prostate. Growth is normally rapid and gives rise to invasion of the pelvic bony walls with

pains radiating into the thighs and legs. The tumours are normally soft and may be gelatinous with a myxosarcomatous look; they may project into the bladder and give rise to the coarsely lobulated polypoid appearances so characteristic of rhabdomyomatous tumours growing beneath a mucous surface. Indeed, it is often difficult to be sure of the primary site of the growth. Khoury and Speer (1944) tabulate 18 cases of prostatic rhabdomyosarcoma but their list is not exhaustive.

I have seen three examples of this tumour in children of 1¾ years, 4 years and 12 years respectively. The oldest child illustrates the fairly typical course of the malady.

A boy aet 12 years was admitted to hospital with retention of urine. The abdomen was swollen and the bladder distended and the prostate was enlarged and hard. Owing to great difficulty in passing, a catheter suprapubic cystostomy was carried out. The patient was transferred to the Western Infirmary. A nodule thought to be an enlarged lymph node was excised from the perineum and was found to be replaced by a cellular malignant growth the exact nature of which was not then appreciated. During the next few months the prostatic tumour extended to fill the pelvis and invaded the rectal wall. Direct extension of the growth into the pelvic bones and ultimately into the whole pelvic girdle followed and death occurred about 10 months after the onset of symptoms.

Post-mortem examination revealed widespread skeletal metastases but secondary growths were not present in the liver or lungs.

Microscopically the primary growth is composed of large cells with rather clear cytoplasm, arranged in a vaguely alveolar pattern. The secondary nodule in the perineum consists partly of short spindle-cells and partly of alveoli of large round cells with hyperchromatic nuclei and scanty cytoplasm. Amongst these occur here and there elongated cells with abundant eosinophilic cytoplasm. The appearances are entirely comparable to the more anaplastic and cellular areas of some of my other cases and I have no doubt that this growth properly belongs to the malignant rhabdomyoma group.

The youngest case, a child aet 1¾ years, was under the care of the late Mr. G. H. Edington, who reported the findings at the Paediatric section of the B.M.A. in 1909 as a myxosarcoma of the prostate. In the subsequent discussion Mr. D'Arcy Power drew attention to the remarkable resemblance of Mr. Edington's case to the polypoid sarcoma of the cervix and vagina in female infants. I have no doubt that Mr. D'Arcy Power's analogy was well founded as this case, like sarcoma botryoides, is a true rhabdomyosarcoma of the prostate.

Myoblastoma of the Urinary Bladder

In 1937 we reviewed the literature on striped muscle tumours of the bladder and since then further cases have been added and the literature again surveyed by Khoury and Speer (1944), and by Hirsch and Gasser (1944). Two cases have recently been reported by Minchin (1947). Since

most vesical tumours tend to become pedunculated the diagnostic signifi-
cance of this feature is less apparent, but the surface of the vesical polyps
is smoother and the processes coarser and more clubbed than in epithelial
tumours, and sometimes myxoma-like nodules of growth are attached to
the bladder wall by filamentous pedicles.

The muscle tumours show two characteristic forms :—

(a) A coarsely lobulated polypoid growth with many bulbous processes
suspended from a common stalk ; such growths generally show unequi-
vocal cross-striations.

(b) A mushroom-shaped tumour with a broad stalk, as is shown in two
of our cases ; these generally lack cross-striations.

The views expressed about the origin of rhabdomyomatous tumours
in the bladder, are either that they arise from myotomic elements carried
down by the Wolffian duct, or from striped muscle which lies in the
posterior superior portion of the prostate in the fœtus and child. No
doubt the latter origin is concerned in the cases arising in the prostate.

Mrs. A. aet 27. Incontinence following birth of the first child. A
pedunculated tumour the size of a nutmeg was found growing just above the
internal urethral meatus, and the whole anterior wall and base of the
bladder were covered by small coarse sessile papillomata. The main mass
was removed and the smaller growths destroyed by coagulating and cut-
ting diathermy. There was no recurrence after six years.

A.E. Male aet 61. Admitted on account of severe painless hæmaturia.
A polypoid ulcerated tumour of roughly mushroom shape arose from the
posterior bladder wall 1½ to two inches above the ureteric bar, the growth
measuring about 2·5 cm. in diameter and 2 cm. in length. The patient
collapsed and died under the anæsthetic.

The So-called Granular-Cell Myoblastoma

This type of growth was described by Abrikossoff (1926) as the pure
myoblastoma and since then it has attracted increasing notice on account
of its comparative frequency, its peculiar histological structure and the
absence of malignant features in most cases. The name myoblastoma is
unfortunate in two respects :—

(a) it suggests a tumour of primitive cells and thus carries an un-
founded suspicion of probable malignancy, and

(b) while the origin of the growth is undoubtedly in striated muscle,
it appears to be derived from adult striated fibres and not from myoblasts.

Good reviews of the general features and topographical distribution of
such tumours have been given by Ringertz (1942), Howe and Warren
(1944), Gray and Gruenfeld (1937) and by Crane and Tremblay (1945),
who have collected 162 cases from the literature including five new ones.
By far the commonest site of this type of growth is the tongue ; next in
frequency come the skin and subctuaneous tissues. The age and topo-
graphical distribution is shown in this table taken from Crane and
Tremblay's paper.

Distribution of Granular-cell Myoblastoma
Crane and Tremblay

Alveolar proc., maxilla and mand.	19
Breast	8
Ear	3
Larynx	8
Lip	4
Muscle	13
Skin	17
Subcutis	17
Tongue	61
Others	12

I have in my collection growths of this character in the tongue (5), skin (2), breast (2), anal region (1) and maxillary alveolar process (1), and I shall illustrate their features from these cases.

Tongue. Granular-cell myoblastoma of the tongue occurs on the dorsal and lateral aspects as a small firm painless nodule paler in colour than the surrounding tongue, often elevated and sometimes almost pedunculated. Over the tumour the squamous epithelium is usually thickened and shows hyperkeratosis. Excision reveals a growth of pinkish-grey colour poorly defined and not encapsulated, the curious abnormal elements that comprise the tumour extending deeply into the muscle bundles of the tongue.

Fig. 11 is from a recently observed male case aet 32, who had noticed a small firm nodule on the anterior part of the tongue. A wedge excision was performed but recurrence took place and two months later the growth had again attained a diameter of 1 cm. Further excision was carried out but it is too early to know whether cure has been achieved.

Case 2. A young woman observed a small painless nodule on the dorsal surface of the tongue near the tip. This was excised with a very small margin, but there has been no further trouble up to almost four years later.

The tumours are composed of large coarsely granular cells, sometimes polygonal or irregularly round, but often elongated in a form generally resembling muscle bundles. The nuclei are small and darkly staining and lie commonly at the periphery of the strand ; the elongated cytoplasmic mass may be syncytial or may show evidence of incomplete sub-division into cells. Each syncytial strand is surrounded by a fine connective tissue sheath, collagenous or reticular. The outstanding feature is that the cytoplasm is composed of innumerable coarse granules between which the cell-substance appears empty. The granules are acidophil and in general present the staining reactions of muscle substance ; but they are much less deeply stained by most of the methods than are the adjacent unaltered muscle fibres. The spread of the tumour cells between and within the

muscle bundles of the tongue leads to pronounced atrophy and disappearance of the normal fibres and their ultimate replacement by the granular cells. The overlying squamous epithelium of the tongue is in many cases thickened and irregular, with hyperkeratosis, elongation of the rete pegs and a downgrowth of epithelial cells very strongly suggestive of squamous epithelioma (fig. 11). The changes are strictly confined to the area of contact between the granular cells and the epidermis, and although cell nests are formed mitoses are very scanty. This epithelial hypertrophy is encountered in similar tumours in other sites, notably in the larynx, but elsewhere not usually so markedly as in the tongue. In spite of the dangerous look of the overlying epithelium, it does not seem to behave as malignant and Ringertz states that neither in his own cases, nor in any reported, has there been recurrence or metastasis of epithelial growth. This is an important point for in some of the reported cases in the larynx and tongue a diagnosis of squamous carcinoma has been made. Clearly if the lesion is benign less drastic surgical measures will suffice.

In the *Skin* an example of granular-cell myoblastoma is seen in a growth from the back of the neck in a man aet 40. It has grown slowly over many months but recently the centre broke down and ulceration occurred. Local excision was performed.

The appearances are quite typical, the granular cells present the characteristic staining reactions. The growth does not seem here to be connected with normal striated muscle as it has arisen in the dermis proper but is spreading into the subcutaneous fat. The overlying squamous epithelium shows the characteristic hyperplasia in marked degree and might be easily mistaken for squamous epithelioma.

In the *Breast* a tumour of similar histological structure was encountered in a woman of 39 years who had complained of pain in the left breast for one year. There was a moderate degree of bilateral hyperplastic cystic disease, but a small solid nodule in the axillary tail of the left breast was found and was excised in course of a simple mastectomy.

The growth measured about 1 cm. in diameter, it was of greyish-yellow colour, was not encapsulated and in general appearance simulated a small scirrhous cancer.

Microscopically the tumour is composed of typical syncytial strands of coarsely granular cytoplasm with fine collagenous stroma between (fig. 12). At the periphery the cells are spreading into the adjacent fat, and foci of lymphocytic infiltration occur mingled with the large granular cells. Epithelial ducts and acini were not seen in any of the sections of the tumour and there is nothing to point to an origin from this source. Gray and Gruenfeld doubt the myoblastic nature of such growths in the breast and consider the appearances in their case suggestive of an origin from the gland acini. It is tempting to speculate that the myo-epithelium of the breast might afford an origin for this growth but there is no histological evidence to support such a hypothesis in my own cases. Similar

tumours have been reported in the breast by Abrikossoff (1931), Meyer (1932), Geschickter (1934), Gray and Gruenfeld (1937) and Simon (1947).

Congenital Epulis. The so-called congenital epulis usually forms a pedunculated mass about 1 cm. in diameter attached to the alveolar process of the maxilla, less often the mandible. It is present at birth, the surface is smooth and of pinkish-grey colour and it may interfere with suckling. Occasionally it may be much larger (Meyer, 1932). The growths are composed of coarsely granular cells resembling those of the granular-cell myoblastoma, and they do not recur after simple excision. Rarely growths of similar structure are met with in adults.

I am indebted to Dr. Robb-Smith for sections and for clinical details of a typical example in the anal region.

A woman aet 28 observed a small lump at the side of the anus of slow growth over a period of seven months, and unattended by pain and discomfort. The growth lay antero-lateral to the anal orifice and posterior to the vaginal wall, and over it the skin was freely movable. In the course of excision the growth was found not be be encapsulated and had to be cut out from the underlying muscles, but it was not attached to the anal sphincter. It measured about one inch in diameter, was of greyish-pink colour and of slightly mucinous feeling. Healing was uneventful and there was no recurrence up to seven years later.

The growth is a typical granular-cell myoblastoma, and shows deep penetration of the muscle bundles by the peculiar granular cells so that one sees normal fibres and long granular syncytial strands lying side by side. A few striated fibres showing partial conversion to granular form were found and the significance of this observation will be discussed later.

Although the granular-cell myoblastomas are usually benign, a small number of unquestionably malignant examples have been reported : in the sacral region with bilateral inguinal lymph node metastases (Geschickter, 1934) ; in the bladder and prostate with generalised metastases (Ravick, Stout and Ravich, 1945) ; in the gluteal region (Ackermann and Phelps, 1946) ; and in the skin with ovarian metastases (Powell, 1946). In our first series (Cappell and Montgomery, 1937), we recorded a malignant myoblastoma of the tongue in which local removal was followed by recurrence and spread to the cervical lymph nodes. The cells in this case were chiefly spindle-shaped and of small ribbon-type without cross striations and they showed abundant fine refractile granules similar to, but smaller than, those of the granular-cell myoblastoma. While this is undoubtedly a malignant myoblastoma, I am hesitant to classify it as a malignant example of the *granular-cell* type.

These examples illustrate the various sites and the usual clinical behaviour of this curious neoplasm, whose histological characters are so unmistakable but of which the fundamental nature is uncertain.

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SURGICAL TUBERCULOSIS OF BOVINE ORIGIN

Lecture delivered at the Royal College of Surgeons of England

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by

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IT IS DIFFICULT to define accurately in these days, just what is meant by " Surgical Tuberculosis." In former times the meaning was clearer, as the term included tuberculosis of bones, joints, abdomen genito-urinary system and various groups of lymphatic glands such as the cervical. Now the surgeon has invaded the province of the physician and is operating on tuberculous disease of the lungs and of the brain, but to compensate for this many tuberculous conditions formerly subject to operation are now treated on more conservative lines. The modern surgical specialist thus requires to have a wide knowledge of the pathology and bacteriology of tuberculosis if he is to understand how the disease arises and how it may progress. The origin of the disease demands study of the two types of infecting bacillus (the human and the bovine) ; the possible ways in which it may progress involves a wide survey of the problems of immunity in tuberculosis which is beyond the scope of the present lecture.

In this country during the last 30 to 40 years the type of bacillary infection in non-pulmonary tuberculosis has changed much. In the earlier years of the present century the bovine type of bacillus was chiefly responsible but recent research, some of the results of which are mentioned in this paper, indicates that the bovine type now plays rather a minor role to its human counterpart. This does not imply that bovine infection in man is no longer a serious problem, for in Scotland alone, with a population of 5,010,400, it has been estimated that 7,939 cases of non-pulmonary tuberculosis were due to the bovine type of bacillus in 1943-44 (Blacklock, 1947).

Historical

Koch discovered the tubercle bacillus in 1882, and it is rather surprising that such an astute observer missed the importance of bovine infection, though as judged from his writings he had undoubtedly met with cases infected with the bovine type of bacillus. Theobald Smith (1896), an American, was the first to differentiate clearly the human from the bovine bacillus. In this country it was Sir Harold Stiles of Edinburgh who first drew attention to the possible importance of bovine infection in human disease, and who stimulated Fraser to investigate the problem. Fraser published his results in 1912 and 1913. Since that time many workers in the field of tuberculosis research, both in this country and abroad, have drawn attention to the part played by the bovine bacillus in human tuberculosis. Such researches have been conducted chiefly in Scotland

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and in Scandinavia, and thus these countries have been credited with a higher percentage of bovine infections than elsewhere. This may not be correct as it may depend on the more extensive investigations which have been carried out in these countries.

This is not the place to discuss the methods involved in the differentiation of the bovine from the human type of bacillus. The bovine grows much less luxuriantly on artificial media, particularly those containing glycerine, than the human and is more virulent for the rabbit.

The Pathway of Infection of the Bovine Bacillus

Infection with the bovine bacillus is derived chiefly, but not solely, from tuberculous cattle, mainly through infected milk. Thus the primary infection is most often in the alimentary system, or the glands draining that system, viz., the cervical, or the mesenteric. Occasionally, however, the pathway of infection is through the respiratory system due to air-borne infection from a patient with open pulmonary tuberculosis caused by the bovine bacillus, or more rarely, as demonstrated by Jensen et al (1940), the infection may be air-borne in the dust of byres housing tuberculous cattle, the dust becoming contaminated with discharges from the animals. From these primary sites of infection the tubercle bacillus may be carried by blood or lymph stream to other tissues such as bone and kidney, there to set up active disease, often many years after infection at the portals of entry. In many cases the lesion at the site of entry of the infection may have healed or become so insignificant that it can be found only with difficulty, if at all, even at post-mortem examination. The interval between primary infection and the development of secondary disease may vary enormously.

There is evidence to show that the tubercle bacillus may long lie latent in a metastatic focus without causing active disease. In this connection the history of a young man, who was under the writer's observation for some years, is of interest. As a child he had invariably consumed pasteurised milk, but when he was 18 he had spent his summer vacation on a farm where he drank large amounts of raw milk from a non-attested herd. About six months later he was operated on for acute appendicitis when a caseous gland was found in the mesentery in relation to the terminal ileum. This was excised and found to contain bovine tubercle bacilli. Milk from the farm was obtained and from it bovine bacilli were isolated. He was in perfect health for the next four years when he developed tuberculous epididymitis on the right side. This was removed surgically and from it bovine bacilli were isolated. A few months later he complained of urinary symptoms and bovine bacilli were found in the urine. Radiological examination showed disease in the right kidney which was removed when a small ulcerative caseous lesion, 2×1 cm., involving one of the calyces was found. From the kidney lesion bovine bacilli were obtained. Tubercle bacilli continued to be excreted in the urine and he died 15 months later from uræmia. At autopsy the left kidney showed moderate tuber-

culosis as also did the prostate, seminal vesicles and the left epididymis : from the kidney lesion bovine bacilli were obtained. There was no evidence of tuberculosis in the mesenteric glands though several of the glands in relation to the terminal ileum were examined both histologically and by guinea-pig inoculation. In this case it would appear that tubercle bacilli from the original lesion in the mesenteric gland had been carried by the blood stream to the right kidney and there had not produced any clinically recognisable disease for a period of at least four years.

The Incidence of Bovine Infection in Various Surgical Conditions

During 1943/44, there was carried out in Scotland an extensive survey of the types of tubercle bacilli responsible for clinical cases of cerebral and of surgical tuberculosis. Many of the figures given in this paper are taken from this survey, a full account of which will be published shortly by the Medical Research Council as a special report.

1. *Alimentary.* As the alimentary system is the main highway followed by the bovine bacillus in the invasion of the human body, it follows that a high proportion of the tuberculous infections in this system will be bovine. In Table I the results of an investigation carried out in Scotland during 1943-44, are shown together with the figures obtained up to 1935.

TABLE I

TYPES OF TUBERCLE BACILLI: ALIMENTARY TUBERCULOSIS: SCOTLAND

Lesions and Dates	−5 yr.		5+ to 15 yr.		Over 15 yr.		Total	
	H.	B.	H.	B.	H.	B.	H.	B.
Abdominal, 1943-44	3	5 (62·5)	1	2 (66·7)	2	4 (66·7)	6	11 (64·7)
Abdominal to 1932 ..	10	51 (83·6)	3	10 (76·9)	2	2 (50·0)	15	63 (80·8)
Cervical Glands, 1943-44	17	30 (63·8)	8	55 (87·3)	27	23 (46·0)	52	108 (67·5)
Cervical Glands to 1935	9	50 (84·7)	13	44 (77·2)	5	2 (28·6)	29*	102* (77·9)
Total Alimentary, 1943-44	20	35 (63·6)	9	57 (86·4)	29	27 (48·2)	58	119 (67·2)
Total Alimentary to 1935	19	101 (84·2)	16	54 (77·1)	7	4 (36·4)	44	165 (78·9)

* 2 Human and 6 Bovine cases included : ages not stated but under 12 years.
*Percentages in brackets.*

In the recent investigation 64·7 per cent. of primary abdominal infections and 67·5 per cent. of tuberculous cervical lymphadenitis were due to infection with the bovine bacillus. The corresponding figures for bovine

infection in abdominal tuberculosis up to 1932 are 80·8 per cent. (Mitchell, 1914b, Wang, 1916-17 ; Blacklock, 1932) and 77·9 per cent. for cervical lymphadenitis up to 1935 (Mitchell, 1914a and b ; Griffith, 1915, 1929 ; Wang, 1916-17 ; Blacklock, 1932 ; Griffith and Smith, 1935). The more recent figures thus show a reduction in the incidence of bovine infection with these types of tuberculosis. In the earlier figures the highest proportion of bovine infections were in the first five years of life, whereas in the 1943-44 investigation the highest incidence was in the over five to 15 years group, suggesting that bovine infection is now occurring later than formerly. This delayed infection may be due to the greater use of dried milk preparations for infant feeding in more recent times, and also to the greater consumption of raw milk by children of the school age. More milk is consumed in the early years of life than at any other and thus there are more opportunities of infection should the milk contain viable tubercle bacilli. Infection of the alimentary system is, however, difficult to produce experimentally in animals by feeding with tubercle bacilli, and the same probably holds for the human subject in whom some coincidental lesion may be an important factor. In childhood, catarrhal inflammatory conditions of the alimentary tract are commoner than in the adult and these may provide the necessary *locus minoris resistentiæ* for the tubercle bacillus to invade the tissues, thus accounting for the greater frequency of bovine infection in the early years of life. In addition, however, there are immunological factors to be considered, for we know that a primary infection with the tubercle bacillus does engender a certain degree of immunity to a subsequent infection. Indeed, this is the basis of B.C.G. vaccination, and if infection with the human type has already occurred such infection may protect against a subsequent infection with the bovine. Thus in the adult, recent primary alimentary infection of bovine origin is relatively infrequent as compared with the child because most adults have already been infected with the human type in the respiratory system. This is clearly demonstrated in Graph 1 showing the percentages of human and bovine infections in surgical tuberculosis at various ages. In the first 10 years of life the proportion of bovine infection was higher than human, but from the fifteenth year the proportion of human infections quickly increased, and from the age of 20 onwards greatly exceeded the bovine.

The usual tuberculous lesions in the alimentary system are in the cervical or mesenteric glands, though careful search may reveal small tuberculous lesions in the tonsils, pharyngeal or intestinal mucosa. The typical textbook ulcers which encircle the bowel are not very common in primary bovine infection, being more frequently secondary to an open lesion in the lung from which tubercle bacilli, in most cases of human type, are discharged and swallowed.

2. *Bones and Joints.* Bone and joint tuberculosis is always secondary to primary infection elsewhere, as for example in the lungs or alimentary system : more often it is in the former. In our recent investigation in

SURGICAL TUBERCULOSIS
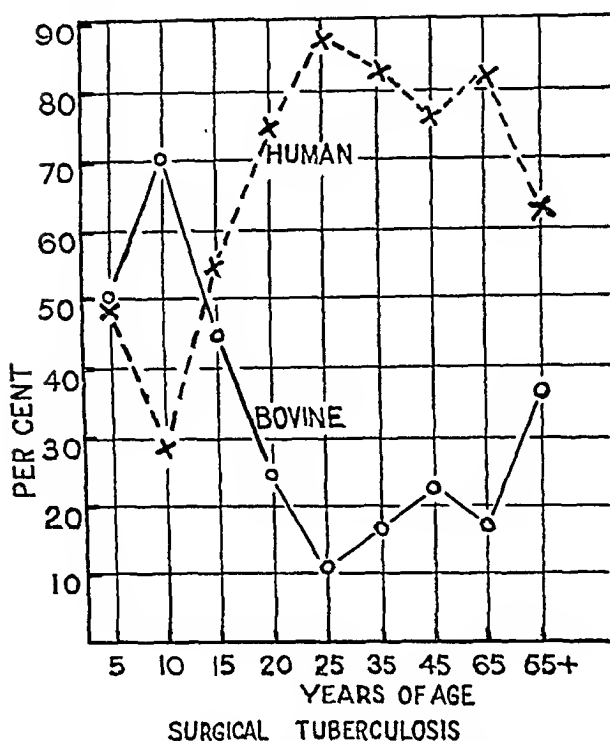
Graph I showing incidence of human and bovine infection in surgical tuberculosis at various ages.

Scotland during 1943-44, the bovine percentage in bone tuberculosis was 12·0 per cent. and in joints 10·1 per cent., or together 11·4 per cent.

TABLE II

TYPES OF TUBERCLE BACILLI: BONE AND JOINT TUBERCULOSIS: SCOTLAND.

Lesion	-5 yr.		5+ to 15 yr.		Over 15 yr.		Total		
	H.	B.	H.	B.	H.	B.	H.	B.	
BONES									
Spine	1	1	8	5	62	5	71	11	(13·4)
Others	10	2	9	1	27	2	46	5	(9·8)
							117	16	(12·0)
JOINTS									
Knee	1	—	7	1	19	1	27	2	(6·9)
Hip	2	1	3	1	13	—	18	2	(10·0)
Others	—	1	1	1	16	1	17	3	(15·0)
							62	7	(10·1)
Total, 1943-44 ..	14	5 (26·3)	28	9 (24·3)	137	9 (6·2)	179	23 (11·4)	
Total to 1935 ..	47*	60* (56·1)	61	30 (33·0)	56	10 (15·2)	164	100 (37·9)	

*. 3 mixed infections with Human and Bovine infections not included.
Percentages in brackets.

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The highest proportion of bovine infection occurred in the first five years of life, falling slightly in the over five to 15 years age group and lowest in those over 15 years. The proportion of bovine infection was slightly higher in tuberculosis of the spine (13·4 per cent.) as compared with that in other bones (9·8 per cent.), and higher in the hip joint (10·0 per cent.) than in the knee (6·9 per cent.). The reason for these different percentages of bovine infection in the various situations is difficult to explain. In the case of the spine Fraser (1929) has shown that the 10th, 11th and 12th dorsal and 1st lumbar vertebræ are more frequently the seat of tuberculosis than any other. He accounts for this by the fact that it is into the large lymphatic field situated at this level that the bulk of the abdominal lymphatics drain. As we have pointed out, the incidence of bovine infection in primary abdominal tuberculosis is very high, varying from 64·7 to 80·8 in different investigations and thus the higher incidence of this type of infection in the spine is possibly due to lymphatic spread from abdominal lesions into the vertebral bodies. Compared with previous work in Scotland between 1912 and 1935 (Fraser, 1912, 1913 ; Wang, 1916-17 ; Griffith, 1916-17 ; 1928, 1929, 1932 ; Munro and Cumming, 1926 ; Griffith and Munro, 1932 ; Blacklock, 1932 ; Griffith and Summers, 1933 ; Griffith and Smith, 1935) the bovine infection in the 1943-44 investigation is less than one-third that previously recorded (37·9) for Scotland, the reduction affecting all age groups.

3. *Genito-Urinary.* From a series of cases of renal tuberculosis investigated during 1943-44, we obtained 28·3 per cent. bovine strains. The bacilli were all isolated from catheter specimens of urine taken during the course of the day and not first thing in the morning. This we have found preferable to and more convenient than examination of a 24 hours' specimen and the results are as reliable.

TABLE III

Types of Tubercle Bacilli : Genito-Urinary Tuberculosis : Scotland

Site	−5 yr.		5+ to 15 yr.		Over 15 yr.		Total	
	H.	B.	H.	B.	H.	B.	H.	B.
Kidney	—	—	1	1 (50·0)	32	12 (27·3)	33	13 (28·3)
Male Generative ..	—	—	—	—	16	6	16	6 (27·3)
Female Generative ..	—	—	—	—	9	2	9	2 (18·2)
Total	—	—	1	1 (50·0)	57	20 (26·0)	58	21 (26·6)
Kidney to 1935 ..	—	—	4	4 (50·0)	42	11 (20·8)	46	15 (21·1)

Percentages in brackets.

Repeated examinations of the urine may be necessary owing to the frequency of intermittent bacilluria, particularly in the incipient phase of renal tuberculosis when the lesions consist of one or more small tuberculous foci, often microscopic in size, in the cortical areas of one or sometimes both kidneys. Such lesions may go on to fully developed tuberculosis or may heal.

Infection with the bovine type was responsible for 27·3 per cent. of cases of tuberculosis of the male generative system (chiefly epididymitis) and 18·2 per cent. of the female (salpingitis or endometritis). In connection with the male generative system the pathway of infection is of some importance as many cases of genital tuberculosis in the male may be due to a descending infection from the kidney *via* the urine from which the infection passes back through the ducts in the prostate, seminal vesicles and vas to the epididymis, often setting up disease in all these structures (Borthwick, 1946).

The almost identical bovine percentage in renal tuberculosis (28.3) and tuberculosis of the male generative system (27·3) found during 1943-44 lends some support to this direct pathway of infection.

In cases of renal tuberculosis investigated in Scotland up to 1935 (Wang, 1916-17; Blacklock, 1932; Griffith, 1932; Band, 1935; Griffith and Smith, 1935), the proportion of bovine infections was 21·1 per cent. (Table III.)

4. *Cerebral.* In tuberculosis of the central nervous system (meningitis or tuberculoma or both) studied during 1943-44 in Scotland, bovine infection was responsible for 11·1 per cent. of the cases, the highest proportion occurring in the first five years of life with 17·9 per cent. As compared with the period up to 1937, this bovine percentage was less than half, the reduction affecting all age groups (Table IV).

TABLE IV

Types of Tubercle Bacilli: Cerebral Tuberculosis: Scotland

Date	−5 yr.		5+ to 15 yr.		Over 15 yr.		Total	
	H.	B.	H.	B.	H.	B.	H.	B.
1943-44	193	42 (17·9)	153	15 (8·9)	152	5 (3·2)	498	62 (11·1)
to 1937	178	72 (28·8)	71	19 (21·1)	30	6 (16·7)	281*	99* (26·1)

Percentages in brackets.   * Included 2 cases, ages not stated.

5. *Pulmonary.* As might be expected the incidence of bovine infection in the lung is low, due to the fact that the air passages are the main route by

which the human bacillus invades the body, chiefly in droplets containing tubercle bacilli coughed up by the patient with open pulmonary tuberculosis. In 166 children up to 13 years suffering from pulmonary tuberculosis we have found the proportions of bovine infections to be 3·6 per cent. Griffith and Munro (1944) in figures collected from various sources found 5·7 per cent. bovine infections in 2,769 cases of pulmonary tuberculosis in adults. The bovine type of bacillus causes the same type of lesions in the lungs as does the human, though from the small number of cases investigated the prognosis appears to be more grave in bovine infections.

The Distribution of Bovine Strains in Town and Country Patients

Considering all the non-pulmonary cases investigated in the 1943-44 investigation in Scotland, we found 13·0 per cent. of the town cases infected with bovine strains and 31·6 per cent. of the country (Table V).

TABLE V

INCIDENCE OF BOVINE INFECTION IN NON-PULMONARY TUBERCULOSIS IN TOWN AND COUNTRY CASES

Site of Disease	Town		Country	
	H.	B.	H.	B.
Abdominal	3	6 (66·7)	3	5 (62·5)
Cervical	28	24 (46·2)	24	84 (77·8)
Bones and Joints*	84	12 (12·5)	94	11 (10·5)
Genito-urinary	28	5 (15·2)	30	16 (34·8)
Cerebral†	306	20 (6·1)	191	42 (18·0)
Total	449	67 (13·0)	342	158 (31·6)

* 1 Human infection, residence not stated.
† 1 Human infection, residence not stated.
Percentages in brackets.

That is, the proportion of bovine infection was almost three times higher in the rural areas as compared with the urban. Of the various surgical

conditions the bovine percentage was much higher in country patients in tuberculosis of the cervical glands, genito-urinary system and brain, whereas it was slightly higher in town patients in abdominal and in bone and joint tuberculosis. It should be noted, however, that the small number of cases in the abdominal group is too small to be significant. -

The cause of this higher incidence of bovine infection in the rural areas as compared with the urban depends on many factors. The first concerns milk supply and this will be first discussed with particular reference to Scotland. The number of attested herds in Scotland is high, there being 4,138 such herds in existence in 1944, representing an increase of 57·6 per cent. over the number in 1939. In populous centres the milk supply is largely in the hands of big dairy combines and they, together with the Milk Marketing Board, adopt high standards for safe milk and also extensively employ heat treatment. These facts must have a great influence on reducing the incidence of bovine infection in urban areas. On the other hand, conditions are different in the rural areas where the producer-retailer is often responsible for the milk supply of a small community and usually safeguards are non-existent. In large centres, however, milks from various herds are subject to bulking which disseminates any infection through large volumes of milk, but at the same time reduces the concentration of that infection which will be destroyed if adequate heat treatment is employed. In the rural areas this bulking of supplies usually does not take place, and thus any infection that may be present is concentrated in a small volume of milk and the consumer, in the absence of efficient heat treatment, is liable to large doses of any infecting organism that may be present. It is these large doses of bacteria, tubercle or any other, which are so liable to overwhelm the natural defences of the body. The smaller burghs in Scotland occupy a position intermediate between the large cities and the rural areas.

All the factors mentioned are reflected in the regional incidence of bovine infection in purely tuberculous surgical conditions (glandular, bone and joint, genito-urinary and abdominal tuberculosis) in Scotland where the bovine percentage in 1943-44 was found to be 34·4. In the large burghs it was 20·1 per cent. and in the rural areas of the counties more than twice as high with 45·4 per cent. (Table VI). In the western region of Scotland where nearly 60 per cent. of the population reside, the bovine percentage in these surgical conditions amounted to 27·8 per cent. In Glasgow, with a population of 1,131,500, the percentage was 8·8 and 13·6 in the large burghs in the Clyde Valley area, and 44·4 in the neighbouring rural counties, from which Glasgow and the large burghs draw their milk supply. In Glasgow, 95 per cent. of the milk sold for human consumption is subject to some form of heat treatment. Thus, in large populous centres like Glasgow, owing largely to safeguards adopted, the incidence of bovine infection is bound to be lower than in the rural areas and the smaller burghs, as in fact was the case.

TABLE VI

PERCENTAGES OF BOVINE INFECTIONS IN SURGICAL TUBERCULOSIS IN (1) SCOTLAND, AND (2) IN WESTERN REGION, ETC.

Area.	H.	B.	Bovine Per Cent.
All Scotland	312	165	34.4
Large Burghs	163	41	20·1
Counties	149	124	45·4
Western Region..	166	64	27·8
Glasgow	62	6	8·8
Large Burghs	38	6	13·6
Counties	66	52	44.4

Sigurdsson (1945) in a masterly survey made in Denmark during the German occupation has also noted the higher proportion of bovine infections in inhabitants of rural areas as compared with urban. Of 165 rural cases of pulmonary tuberculosis or pleurisy he found that 40·6 per cent. were infected with the bovine type, whereas this type of infection was found in only 3·6 per cent. of 362 urban cases. He also pointed out that 94·5 per cent. of the bovine infected cases had been in contact with tuberculous cattle. As he had demonstrated living tubercle bacilli in the dust and dirt of byres and stables, he was of the opinion that in many cases the pulmonary disease was due to the inhalation of such dust.

The increased precautions which are now adopted with regard to the milk supply, as compared with those in the past when town byres existed, is reflected in the reduction in the proportion of bovine infections in the 1943-44 Scottish investigation, as compared with those reported from 1912 onwards ; some of these decreases have already been commented on above. But to summarise ; we find that up to 1937, the percentage of bovine infections in surgical tuberculosis in Scotland was 53·6 (260 human and 300 bovine), whereas in 1943-44 the percentage had fallen to 34·4. With further precautions regarding the milk supply and the gradual elimination of tuberculous cattle, one may hope in the future to see the complete elimination of surgical tuberculosis, due to the bovine type of bacillus, though there will still remain the major problem of that due to the human type.

Another factor influencing the proportion of bovine infection in any community is the extent of infection with the human type. It would appear that as in syphilis so long as living organisms are present in the body, whether producing active disease or not, a subsequent exogenous reinfection is uncommon. It is not possible, at present, to explain satis-

factorily this peculiar immunological state which does not appear to depend, as in the case of many other bacterial infections, on humoral antibodies. With the greater density of population in towns, the urban dweller is more liable to infection with the human type than the rural, and such infection will protect, though not absolutely, against a subsequent bovine infection. In support of this is the rarity of mixed infections with the two types of bacilli.

The Pathology of Bovine Infection in Man

On the pathological side it is not possible, at present, to decide from the morbid anatomical or the histological evidence in a case whether a lesion is due to human or bovine infection. This is because so many variable factors are involved, such as the natural and acquired immunity and the degree of hypersensitivity of the subject, the dose and the virulence of the infecting organism. All these variable factors influence the morbid anatomical findings, and with the right combination of these a bovine infection can be, and frequently is, as severe as that due to the human type.

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"OBSERVABLES" AT THE ROYAL COLLEGE OF SURGEONS
1. WOOLNER'S "PUCK"

"AMONG OTHER OBSERVABLES at Chirurgeons' Hall," to use Samuel Pepys' phrase of another time, is a bronze figure of "Puck" by Thomas Woolner, R.A. Both the statuette and the sculptor are of interest to surgeons and naturalists.

Woolner, who was born in 1825, began to model his figure of "Puck" when 19 years' old and the finished work in plaster was exhibited at the British Institution in 1847. It is one of the most admired and attractive of Woolner's early works. The little figure was cast in bronze for Louisa Lady Ashburton in 1866 and exhibited in the Royal Academy. In 1908, after many years of persuasion, Woolner's widow, who feared that the original model might be damaged, allowed another cast to be made for Sir John Bland-Sutton, who bequeathed it to the Royal College of Surgeons where it has an honoured place in the Entrance Hall.

While Woolner was modelling "Puck" he noticed the little pointed tips that are seen in so many human ears, and exaggerated those in the statuette to make the ears fawn-like in character, thus giving the little fellow an impish look. When Darwin sat to Woolner in 1868 for a Medallion Portrait for reproduction by Wedgwood the artist imparted to the great naturalist his ideas on his discovery, and later sent Darwin a drawing of one of his models of a human ear showing a well-marked example of what is generally known as "Darwin's Tubercle." It is, however, interesting to find that Darwin gives credit to Woolner for the observation, for, in acknowledging the drawing, he wrote from Down House in 1869 :—

My dear Woolner,

Very many thanks for the drawing: it does excellently. The "Woolnerian tip" is worth anything to me.

<div align="center">Yours sincerely,
Ch. Darwin.</div>

In 1871, the year that "The Descent of Man" was published, Darwin referred to the matter again in another letter to Woolner. I quote the letter in full because the first paragraph is also of interest as an amusing illustration of the inquisitive turn of mind of the seeker after truth.

My dear Woolner,

I daresay you often meet and know well painters. Could you persuade some *trustworthy* men to observe young and inexperienced girls who serve as models, *and who at first blush much,* how low down the body the blush extends ? . . . Moreau says a celebrated French painter once saw a new model blushing all over her body. So that I want much to hear what the experience is of cautious and careful English artists. I always distrust memory—can you aid me ?

The tips of the ears have become quite celebrated. One reviewer (Nature) says they ought to be called, as I suggested in joke, Angulus Woolnerianus.

"PUCK" BY THOMAS WOOLNER, R.A.

A great German physiologist is very proud to find that he has the tips well developed and I believe will send me a photograph of his ears ; and if a good case, I think I would have it photographed on wood engraved for new Edit. Making of course no change in my text.

Yours very sincerely,

Ch. Darwin.

One might think from this letter that Darwin was cooling in his desire, to give credit to Woolner, but the following extract from " The Descent of Man " makes it clear that he wished his recognition of Woolner's observation to be on permanent record.

" The celebrated sculptor, Mr. Woolner, informs me of one little peculiarity in the external ear, which he has often observed both in men and women, and of which he perceived the full significance. His attention was first called to the subject whilst at work on his figure of " Puck," to which he had given pointed ears. He was thus led to examine the ears of various monkeys, and subsequently more carefully those of man. The peculiarity consists in a little blunt point, projecting from the inwardly folded margin, or helix . . ."

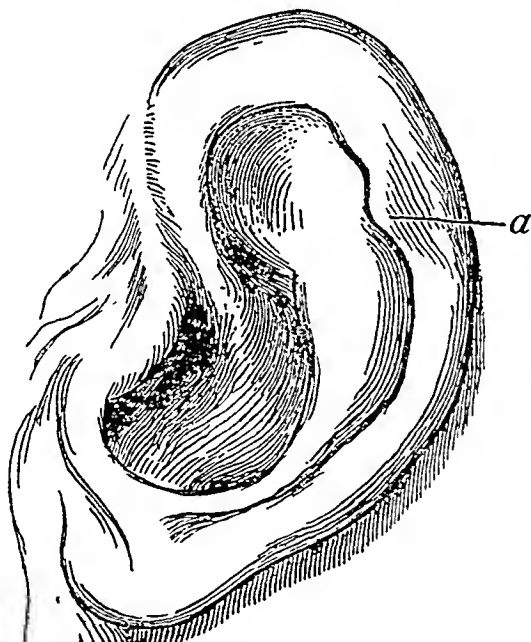" Mr. Woolner made an exact model of one such case, and sent me the accompanying drawing . . ."

Fig. 2—Human ear, modelled and drawn by Mr. Woolner. (a) The projecting point.

" The following woodcut is an accurate copy of a photograph of the foetus of an orang, in which it may be seen how different the pointed outline of the ear is at this period from its adult condition, when it bears a close general resemblance to that of man."

Fig. 3—Fœtus of an Orang. Exact copy of a photograph, showing the form of the ear at this early stage.

" It is evident that the folding over of the tip of such an ear, unless it changed greatly during its further development, would give rise to a point projecting inwards. On the whole, it still seems to me probable that the points in question are in some cases, both in man and apes, vestiges of a former condition."

Woolner, in 1870, sculptured a marble bust of Darwin in addition to the Wedgwood Medallion, and there is a plaster cast of this in the Royal College of Surgeons. He executed many other portrait busts of medical and surgical interest :—William Hey in Leeds Infirmary, De Morgan in The Middlesex Hospital, Sir William Gull, Bence Jones in the Royal Institution, Sir John Simon and Richard Quain in the Royal College of Surgeons, Huxley, of which there is a plaster cast in the College, and the bust of John Hunter in Leicester Square, of which a photograph appeared in THE ANNALS of November, 1947.

One further word about the artist. Woolner was a man of many friends among whom he numbered Rossetti, Ford Madox Brown, Tennyson, Carlyle, Browning and Darwin. He was disappointed when, after executing a memorial tablet with a Medallion Portrait of Wordsworth for Grasmere Church, his design for the Wordsworth Monument was not accepted, and he determined to leave England. Like so many at that time he felt a desire to try his luck at the Australian goldfields, in order, as he hoped, to get money to carry out his ideas in sculpture, and do ideal works. He sailed from Plymouth on July 24th, 1852, by the *Windsor*, and reached Port Philip, Victoria, on October 27th.

Woolner spent two years in Australia. He was not successful in his mining expeditions and, as he wrote to Mrs. Tennyson, " did not find nearly enough gold to pay the expenses necessary : but I by no means regret having visited the magnificent country, some of the scenes in which gave me more pleasure than any I ever saw before, being more

wild, vast and solitary." In the later part of his stay in Melbourne and Sydney he executed 24 Medallion portraits (most of which were cast in bronze), and came home in the hope of securing the commission for a statue of W. C. Wentworth. This he failed to get, but notable examples of his work are to be found in Australasia—Tennyson and Sir Thomas Elder in Adelaide, Godley in New Zealand, and Earl Russell, the Earl of Derby and the colossal statue of Captain Cook in Sydney. A. W.-J.

ANOTHER "OBSERVABLE" AT THE ROYAL COLLEGE OF SURGEONS

2. COPY OF STATUETTE OF WILLIAM HARVEY

Copy of Statuette by C. B. BIRCH, A.R.A.

Made in 1888 to celebrate the 260th anniversary of the publication of
" Exercitatio anatomica de motu cordis et sanguinis in animalibus."

DIARY FOR FEBRUARY (16th—27th)

Mon. 16   5.00   SIR HAROLD GILLIES—Skin Flaps : Indications and Technique.
          5.00   DR. E. W. FISH—Gingivectomy.
          6.15   DR. J. F. BROMLEY—Radiotherapy in Tumours of the Mouth.
Tues. 17   5.00   PROF. M. C. WILKINSON—Hunterian Lecture—Some Observations on the Pathogenesis and Treatment of Skeletal Tuberculosis.*
  —   5.00   MR. T. WARD—Surgery in Relation to Prosthesis.
Wed. 18   5.00   MR. A. B. WALLACE—Free Skin Grafting—Methods and Application.
          5.00   SIR ALEXANDER FLEMING—Penicillin.
          6.15   PROF. H. STOBIE—Role of Dental Surgery in Medicine.
Thur. 19   5.00   PROF. J. BEATTIE—Bernhard Baron Lecture—Hormonal Changes after Injury.*
          5.00   MR. S. H. WASS—Osteomyelitis of Mandible.
          6.15   DR. S. BLACKMAN—Radiology.
Fri.   20   5.00   MR. R. P. OSBORNE—Burns and Their Early Treatment:
          5.00   MR. H. T. ROPER HALL—Therapeutics in Dentistry.
Mon. 23   5.00   PROF. T. POMFRET KILNER—Cleft Lip and Palate Repair.
          5.00   MR. P. R. SHEPHERD—Fractures of the Mandible (I).
          6.15   MR. F. C. ORMEROD—Diseases of Maxillary Sinuses.
Tues. 24   5.00   MR. P. H. JAYES—Fractures of the Facial Skeleton.
          5.00   MR. P. R. SHEPHERD—Fractures of the Mandible (II).
          6.15   MR. A. McLEOD—Posterior Restorations (I).
Wed. 25   5.00   MR. J. N. BARRON—Hand Injuries.
          5.00   MR. V. ZACHARY COPE—Actinomycosis.
          6.15   MR. B. W. FICKLING—Replacement of Tissue Loss in Jaws.
Thur. 26   5.00   MR. J. B. CUTHBERT—Hand Deformities : Reparative Surgery.
          5.00   PROF. F. H. BENTLEY—The Uses and Abuses of Drugs in Surgery.
          6.15   MR. W. KELSEY FRY—Impacted Third Molar.
Fri.   27   5.00   SIR ARCHIBALD McINDOE—External Genitalia : Treatment of Congenital Deformities.
          5.00   SIR CECIL WAKELEY—Mandibular Joint.
          6.15   MR. H. L. HARDWICK—Gingivitis of Pregnancy.

DIARY FOR MARCH

Mon. 1   5.00   MR. R. J. McNEILL LOVE—Erasmus Wilson Demonstration—The Biliary Apparatus.*
        5.00   SIR ARCHIBALD McINDOE—Fractures of the Middle Third of the Face.
        6.15   PROF. W. E. HERBERT—Anterior Restorations.
Tues. 2   5.00   MR. B. W. FICKLING—Epithelial Inlays.
        6.15   MR. A. C. McLEOD—Posterior Restorations (II).
Wed. 3   5.00   MR. P. H. MITCHINER—Erasmus Wilson Demonstration—Enlargement of the Testes and Epididymis.*
        5.00   MR. L. E. C. NORBURY—Diseases of the Salivary Glands.
        6.15   PROF. W. E. HERBERT—Periapical Infection.
Thur. 4   Pre-Medical Examination begins.
        5.00   SIR HAROLD GILLIES—Cleft Palate.
Fri.   5   5.00   DR. L. W. PROGER—Erasmus Wilson Demonstration—Giant Cells.*
        5.00   PROF. F. C. WILKINSON—Complications following Extractions.
Mon. 8   5.00   DR. L. W. PROGER—Erasmus Wilson Demonstration—Giant Cells.*

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* Not part of courses.

Wed. 10	5.00	MR. R. W. RAVEN—Erasmus Wilson Demonstration—The Surgical Manifestations of Boeck's Sarcoid.*
	7.00	Monthly Dinner for Fellows, Members and Licentiates.
Thur. 11		First Membership and D.T.M. & H. Examinations begin.
	5.00	MR. V. ZACHARY COPE—Erasmus Wilson Demonstration—Varieties of Intestinal Obstruction.*
Fri. 12		D.C.H. Examination begins.
	5.00	MR. LEON GILLIS—Arris and Gale Lecture—Recent Advances in the Treatment of Arm Amputations, Cineplastic Surgery and Arm Prostheses.*
Mon. 15	5.00	DR. E. M. DARMADY—Arris and Gale Lecture—Acute Uræmia ; its Ætiology and Basis for Treatment.*
Tues. 16	5.00	MR. A. J. GARDHAM—Surgery of Malignant Disease of the Pharynx.
Wed. 17	5.00	PROF. LAMBERT ROGERS—Surgery of the Spinal Cord.
Thur. 18	5.00	MR. RODNEY MAINGOT—Surgery of Peptic Ulcer.
Fri. 19		L.D.S. Examination begins.
	5.00	MR. A. DICKSON WRIGHT—Varicose Veins and Ulcers.
Mon. 22	5.00	SIR REGINALD WATSON-JONES—Fractures of the Spine.
Tues. 23		Date of Council Election announced.
	3.45	MR. R. J. LAST—Arnott Demonstration—The Kidneys.*
	5.00	MR. M. F. NICHOLLS—Surgery of the Urethra and Bladder.
Wed. 24	3.45	MR. R. J. LAST—Arnott Demonstration—The Pectoral Girdle.*
	5.00	MR. A. HEDLEY WHYTE—Surgery of the Rectum.
Thur. 25	3.45	MR. R. J. LAST—Arnott Demonstration—The Knee Joint.*
	5.00	MR. T. TWISTINGTON HIGGINS—Urinary Obstruction in Childhood.
Fri. 26		College closed.
Mon. 29		College closed.
Tues. 30	5.00	MR. R. M. HANDFIELD-JONES—Some less usual examples of Acute Intestinal Obstruction.
Wed. 31	5.00	PROF. G. GREY TURNER—Surgery of the Spleen.

* Not part of courses.

THE MANDIBULAR JOINT

Lecture delivered at the Royal College of Surgeons of England

on

27th February, 1948

by

Sir Cecil Wakeley, K.B.E., C.B., D.Sc., F.R.C.S.

Vice-President Royal College of Surgeons of England,

Senior Surgeon King's College Hospital .

and

West End Hospital for Nervous Diseases

THE MANDIBULAR JOINT is a synovial joint of the hinge type. It is a somewhat complex structure as it contains an articular disc which divides the joint into two synovial cavities. The joint is a stable one and dislocation of the joint itself or of its meniscus or articular disc is uncommon.

The articular disc is a dome-like structure very closely applied to the convexity of the mandibular condyle. As far as thickness is concerned it is irregular, being very much thicker in its centre and anteriorly; between these two thickenings there is a distinct depression (Fig. 1). The posterior part of the cartilage is very thin and passes well down over the posterior surface of the condyle and fuses behind with the capsule. I have examined over 100 specimens of the articular disc in the anatomical department of King's College and in the post-mortem department at King's College Hospital and all conform to the description I have given.

This description is, however, at variance with the descriptions given in the current standard text-books of anatomy, the most common description being that the articular disc is an oval plate thinnest at the centre, where it is occasionally perforated, and thickest posteriorly. I have never seen a perforated articular disc in this joint in either young or old subjects. The anatomical description to which most of the text-books conform must have been handed down for generations and nobody has troubled to investigate the matter and verify the commonly accepted description. Most text-books of anatomy contain quite good sectional diagrams of this joint, which, to the most casual reader, are obviously at variance with the text description.

The mandibular joint is divided by the articular disc into two separate cavities, each provided with a distinct synovial membrane. The circumfer-

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ence of the disc is adherent to the capsular ligament and anteriorly affords partial insertion to the external pterygoid muscle. It is owing to the fact that there are two definite cavities in this joint that movements of two different kinds can take place. These movements are somewhat complex. Gliding movements, whereby the articular disc and the condyle move together as one on the temporal bone, take place in the upper synovial cavity. Further, a rotation between the disc and the condyle occurs in the lower synovial cavity. Probably these two movements rarely occur independently of one another.

Displacement of Articular Disc

The displacement of the articular disc is an uncommon condition and et it is one which every surgeon or dental surgeon should not only be amilar but should be ready to deal with.

The somewhat frequent displacement of the medial meniscus if the knee-joint is due to the fact that the cartilage is placed at the peri-hery of a very shallow concavity, and also to the fact that the internal ateral ligament of the knee-joint is adherent to it. The latter fact is mportant, since whenever this ligament is put on the stretch it tends o pull the meniscus beyond the limits of the joint. Displacement of he internal meniscus of the knee-joint is the result of some sudden movenent of the joint; similarly, displacement of the mandibular disc or neniscus is due to some sudden or irregular contraction of the external pterygoid muscle. Unlike the medial meniscus of the knee-joint, which may be torn or ruptured, the mandibular disc always remains intact. Displacement of the cartilage may be caused by a violent cough, sneeze, or yawn, the external pterygoid muscle contracting during the rapid closing of the mouth and drawing the articular disc obliquely forwards and inwards. Occasionally, displacement may result from a blow on the jaw when the mouth is open, the trauma not being of sufficient force as to cause dislocation of the condyle over the eminentia articularis of the temporal bone. Again the displacement occurred in two of my patients as a result of extraction of the lower molar teeth.

The posterior thinned-out part of the disc becomes detached from the capsule, and this permits it to be drawn forwards and inwards by the external pterygoid muscle, and as the mouth closes by elevation of the jaw the disc of cartilage becomes severely crushed between the condyle and the temporal bone (Fig. 2).

The symptoms caused by such a displacement are often very characteristic. The patient experiences sudden acute pain in the joint which may be referred to the pinna or the skin above the pinna. This referred pain is due to the fact that the auriculo-temporal nerve not only supplies the joint but also gives off sensory branches which supply the upper part of

the pinna and the skin above it. All attempts to close the mouth are painful and the patient will sometimes volunteer the remark " there is something sticking in front of my jaw which prevents my shutting my mouth ! "

Excessive salivation is often a marked feature, but in old-standing cases, or in those in which the disc is very mobile owing to repeated displacement, it is often so slight as not to cause the patient any annoyance. Mastication is attended with considerable difficulty, as slight chewing causes acute pain. This, however, becomes less marked in a day or so, probably due to the fact that a definite synovitis is set up when the primary displacement takes place. This synovitis causes the capsule of the joint to become very tense, and this in itself will account for the pain felt when movements occur in the joint. When the synovitis becomes chronic the pain becomes much less marked and greater mobility of the disc becomes possible. Quite often the patient suffers from continual recurring displacement which cannot be regarded as acutely painful, but it is very distressing and out of position and causes a definite audible snap. In not a few cases I have seen, this snap has been a source of great annoyance to the patient, especially at meal times, the patient being afraid to eat in public for fear of attracting attention.

As regards treatment, reduction is much more likely to succeed and be permanent if it is performed on the occasion when the disc is first displaced. In old-standing cases the effect of reduction is only temporary. The best method of reduction is to keep up continual pressure behind the condyle of the jaw while the mouth is open. By this means the convex upper part of the condyle will be insinuated into the concave lower surface of the articular disc. After a few minutes of this continued pressure the mouth is closed by slowly elevating the jaw. Sometimes the disc slips back with an audible click, while in others nothing is noticed at all, but the patient states that the obstruction in the joint has disappeared. Several attempts at reduction are often necessary before reduction is complete.

In cases where displacement is more or less continuous the same changes take place in this joint as occur in the larger joints—e.g., the knee when recurrent displacement of the medial meniscus takes place. The peri-articular tissues, capsule, ligaments, and muscles become relaxed ; this allows the disc to become displaced whenever the mouth is widely opened. In these chronic cases no treatment, with the exception of excision, is of any avail. Fortunately, most of these patients are quite content to put up with the slight pain and the inconvenience caused by the repeated " snapping " of the disc. Occasionally, however, one meets with a patient who implores the surgeon to give relief from the chronic pain and the continual " snapping " which is described as unbearable. One of my patients was actually contemplating suicide unless relief from the noise could be obtained. She explained that she was forced to take her meals by herself as her husband objected to the noise she made when she was eating.
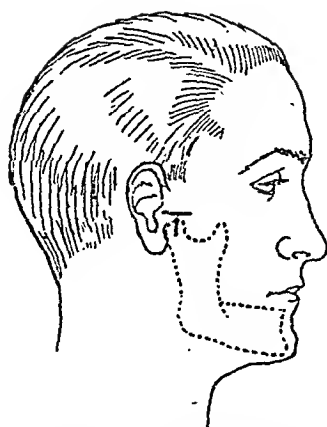
Fig. 3

Out of the thirty-two cases who have come to me for treatment with recurrent displace- of the articular disc, operation was only considered necessary in twelve.

The operation is often difficult because of the small size of the joint. The mandibular joint is opened through a T-shaped incision (Fig. 3). A large incision cannot be made owing to the important structures which come into relation with the capsule. The superficial temporal artery and the auriculo-temporal nerve are immediately behind and the upper branch of the facial nerve crosses the joint on its external surface (Fig. 4).

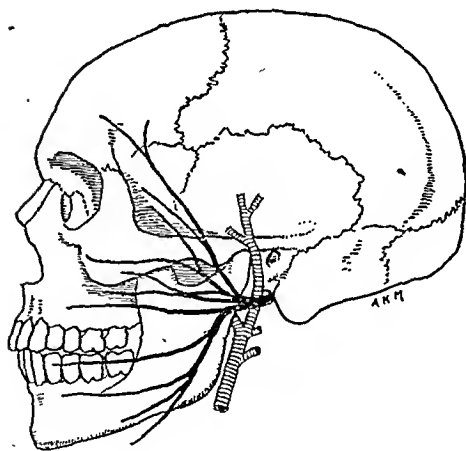The capsule is opened and its edges retracted, the mandible is then depressed

Fig. 4

Fig. 5

The immediate relationships of the mandibular joint. The superficial temporal artery passes behind the joint, while branches of the facial nerve pass just in front.
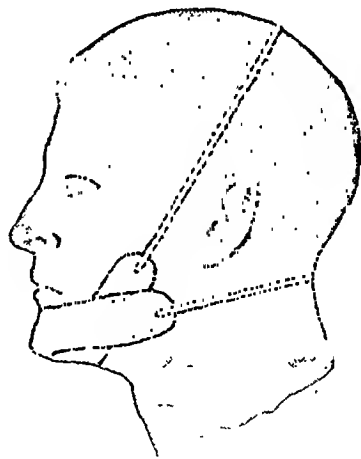
A four-tail bandage made of supple leather is an excellent way of keeping the joint immobilised.

by the anæsthetist and the torn articular disc comes into view; it is grasped by a small pair of artery forceps and delivered into the wound. The anterior attachment of the disc is severed by means of a tenotomy knife. The opening in the capsule is closed by means of interrupted catgut sutures. The skin is approximated by three silkworm sutures.

For the first twelve hours after the operation the mouth is kept closed by means of a four-tail bandage (Fig. 5). After this an elastic strap is worn so that the mouth can only be opened slightly. At the end of the week the stitches are removed and a collodion dressing applied. The patient only requires to be in bed for the first post-operative day. This type of operation has given very satisfactory results.

Dislocation of the Mandible

The temporo-mandibular joint is quite a stable structure and is well protected, so that dislocation is not common. Forward dislocation may result from muscular action, or from trauma as in the case of a blow received on the point of the chin when the mouth is open. It may follow the forcible extraction of one of the lower molar teeth or the use of an elevator in removing a stump from the posterior part of the alveolus. Dislocation may occur during a fit of laughing, gaping, or violent yawning, or when an attempt is made to take too large a bite. In other cases dislocation has resulted from the excessive use of a dental prop or a gag, or from forcing too large a body into the mouth during anæsthesia.

The mechanism of the dislocation is quite simple—the condyle of the mandible slips over the eminentia articularis into the zygomatic fossa (Fig. 6). The inter-articular fibro-cartilage follows the condyle, as the external pterygoid muscle is attached to both. The dislocation is usually bilateral but may be unilateral, depending on the exciting cause. The mouth is held open and the interval between the teeth is about one inch. The lower jaw is fixed by the contraction of the muscles surrounding the joint, and it is this muscle spasm which causes the pain in this dislocation. Speech and deglutition are impaired, and saliva continuously dribbles over the lip. On examination a hollow can be palpated immediately in front of the tragus where the condyle is normally situated, and in front of this the condyle itself can be felt. On passing a
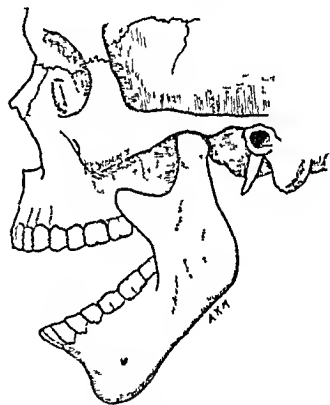

Fig. 6

Anterior dislocation of the mandible, the condyle has passed beyond the eminentia articularis and cannot be replaced by muscular action alone.

finger into the mouth, the coronoid process can be found in an abnormal position just below the zygoma.

The treatment consists in reduction of the dislocation at the earliest possible moment. As a rule, reduction is quite easy, as all that is necessary is to depress the condyle of the jaw below the level of the eminentia articularis, when the contraction of the posterior fibres of the temporal muscles, the masseter, and the internal pterygoid muscles quickly pulls it back into the glenoid cavity. It is necessary for the surgeon to protect his thumbs with a towel, as during reduction the contraction of the power-

ful muscles of mastication may result in a severe injury when the upper and lower teeth come into contact. With the thumbs so protected the surgeon faces the patient, who is seated in a chair. Downward pressure is made by the thumbs on the lower molar teeth, until the condyle slips backwards over the eminentia articularis. It is rare for this reduction to fail, but should it do so, easy reduction can be obtained under a general anæsthetic. After reduction the jaw is kept at rest by the use of a barrel bandage which should be kept on for ten days to a fortnight (Figs. 7 and 8).
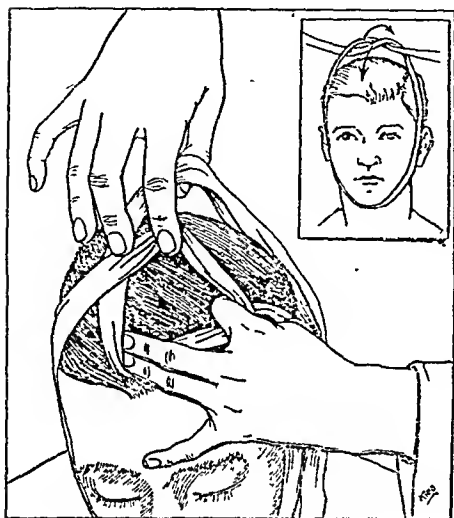

Fig. 7

The application of the barrel bandage. A two-inch bandage is passed under the lower jaw and tied in a simple knot over the vertex. The knot is then opened, and one loop is slipped over the forehead, and the other over the occiput.
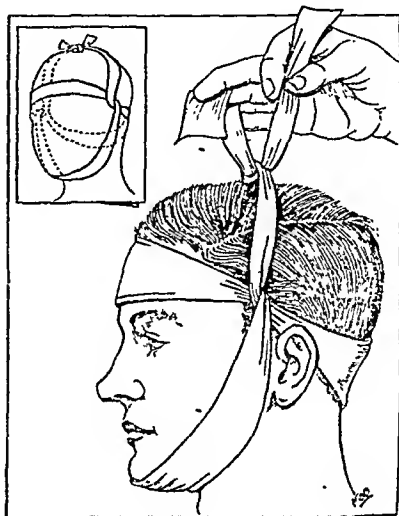
Fig. 8

The completion of the barrel bandage. The twist in the bandage on one side comes to lie just above the ear, while on the other side the longer end of the bandage is passed under the part encircling the head, and the two ends are tied over the vertex.

As a rule, dislocation at this joint does not weaken the articular and peri-articular structures, so that recurrent dislocations are rarely seen. However, exceptional cases do occur when the joint is quite lax owing to repeated dislocation having taken place, the patients often having surprisingly little pain or discomfort and being able to reduce the luxation quite easily themselves.

Unreduced dislocations may be seen occasionally owing to the fact that the condition has not been diagnosed. A false joint may form and, gradually, the patient manages to eat, swallow and talk, but the dislocation still remains very obvious. If the dislocation has been present for some months, reduction may be impossible owing to the fibrosis of some of the masticatory muscles. In such cases it is advisable to excise the condyle to allow of better movement of the jaw and to restore normal mastication.

Dislocation of the jaw backwards is always associated with fracture of the tympanic plate and separation of the cartilage of the auricle, while dislocation upwards results in a fracture of the middle fossa of the skull ; both these fracture-dislocations are uncommon and are usually fatal accidents.

Synovitis and Arthritis of the Temporo-Mandibular Joint

Acute Synovitis. This is uncommon, but may be seen during an attack of acute rheumatism. There is some swelling of the articular and peri-articular structures, due to synovial effusion. Movements of the joint are painful and the patient is often afraid to speak or open the mouth. In most cases complete resolution takes place, but sometimes intra-articular adhesions form which eventually lead to impairment of movement.

Serum synovitis occasionally occurs in this joint, but leaves no after-math.

Chronic Synovitis. This is a rare condition, but may be seen in cases of recurrent subluxation of the joint.

Acute Arthritis of the joint may be pyæmic in origin when it is a sequelæ of one of the exanthemata, or it may be secondary to gonorrhœa, acute parotitis, or acute otitis media. Post-scarlatinal otitis media is quite likely to be the cause of direct extension of inflammation through the tympanic plate into the joint. In children it may be difficult to diagnose the condition when an acute otitis is present, and an abscess formation, which rapidly points and bursts externally, may be the first indication that the joint is infected. It is important that such an abscess should be incised and drained as soon as it is diagnosed. Acute arthritis is invariably followed by ankylosis, which requires excision of the condyle at a later date when all signs of inflammation have subsided.

The use of penicillin and the sulphonamide group of drugs has given excellent results in these cases and ankylosis may be avoided.

Chronic Arthritis. Osteo-arthritis is by no means rare in this joint, although it is quite often overlooked as a cause of pain in the joint. With modern methods of radiology the condition can be seen quite easily in a skiagram ; it may be symmetrical, and is characterised by considerable enlargement of the condyle of the jaw which causes it to bulge laterally so that it can be felt in front of the tragus of the ear. Movements of the joint are painful and limited, and crepitus can nearly always be elicited. If the condition is bilateral, the lower jaw appears to be pushed forward, rendering the chin quite prominent ; if, however, it is unilateral, the jaw becomes deflected to the sound side. Loose bodies may form in some cases and may cause " locking " of the joint, while in others there is extensive lipping of the periphery of the condylar cartilage, proliferation of the synovial villi, and " lipoma arborescens " may be in evidence. As the disease progresses the inter-articular cartilage may disappear

completely, and the glenoid cavity, as it enlarges, may assume a flattened outline so that there may even be a partial dislocation. If pain and limitation of movement are complained of, the only satisfactory treatment is excision of the condyle, as all other forms of treatment are useless and a waste of time.

The operation of excision of the condyle of the jaw is carried out through a curvilinear incision, commencing over the middle of the zygoma, and passing downwards in front of the tragus. In this operation the surgeon is working in a somewhat cramped space with the zygoma above, the facial nerve below, the parotid gland in front, and the external ear behind. After the skin and subcutaneous tissues have been incised, the small flap is turned upwards. A transverse incision is now made at the posterior end of the zygoma, opening up the capsule of the joint and the synovial membrane. The neck of the mandible is exposed, and a fine Gigli saw is passed round this with a small aneurysm needle. The neck of the bone is cut through with this saw and the condyle removed. Sometimes the neck of bone may be divided with a small pair of cutting pliers. Any, bleeding from the cut surface of the bone can be controlled by the firm application of some bone wax to the raw area. A small piece of celluloid can be placed over the cut surface of the bone and retained with a stitch. This prevents any chance of ankylosis of the joint. A piece of fascia may perform the same purpose, while some surgeons advocate the use of a portion of the tunica vaginalis testis. The wound is closed with two or three interrupted sutures, no drainage being employed. The patient is encouraged to move the joint at an early date. The results of such an operation as this are really gratifying, and patients are quite pleased.

Tuberculous Arthritis. It is often difficult to be certain whether tuberculous disease in this joint has arisen in the bone or in the synovial membrane. The affection itself is rare and is only seen when the disease is fairly well advanced. It runs the usual course of any tuberculous joint affection and ends in caries of the condyle. Secondary infection may occur, leading to ankylosis. The condition is very chronic and has been mistaken for osteo-arthritis on more than one occasion, the true diagnosis only being made when excision of the condyle was performed and the bone subjected to microscopical examination. Excision of the condyle and thorough cleansing of the cavity of the joint, followed by the application of B.I.P.P., is the treatment for this condition.

Neuropathic arthritis of this joint is a very rare entity.

Fixity of the Jaw (Trismus)

Immobility or closure of the jaw may be the result of a great variety of conditions. The following are the most important :

(1) Fibrous or osseous ankylosis may result from any acute suppurative condition (Fig. 9).

(2) Old-standing dislocations, where a false joint has formed in front of the glenoid cavity ; in chronic cases of osteo-arthritis where the amount

of osteo-arthritic outgrowth is excessive ; in cases of fracture of the neck of the mandible with excessive callus formation ; and, rarely, in cases of cyst, or tumour formation in the head or neck of the mandible.

(3) Cicatricial contraction of the surrounding soft structures, as from burns and scalds, lupus, cancrum oris, and scars resulting from operations or the application of radium in the pterygoid region. Gummatous infiltration of the masseter muscle leading to fibrosis occasionally occurs as a result of syphilis. Very rarely, myositis ossificans has been the cause.

(4) Spasm of the muscles of the jaw due to reflex irritation or impacted third molar tooth. Occasionally, the spasms may be hysterical in nature, and are one of the early signs of tetanus.

(5) The pain and swelling of local inflammatory conditions · often render opening of the mouth impossible ; among the commoner of these may be mentioned mumps, parotid abscess, lymphadenitis, acute tonsilitis, and acute alveolar periostitis. Any malignant growth of the face or cheek may seriously impair the mobility of the jaw. Likewise, extensive actinomycosis of the skin covering the jaw, and acute necrosis of the mandible with sinus formation may lead to extensive fibrosis which will eventually cause fixity of the jaw.

The treatment of the different varieties of fixation of this joint will of necessity vary according to the causative conditions.

Fibrous ankylosis can be dealt with by excision of the condyle of the jaw already described.

Bony ankylosis often presents a difficult problem owing to the fact that the surgeon is working in a limited space, and any attempt to remove the neck or head of the condyle by means of a chisel or osteotome may result in a fracture into the middle ear or injury to the facial nerve. A fine pair of nibbling forceps may be used, and the bone in the region of the neck of the mandible be carefully removed. piece by piece, until the bone is completely divided. It is important to remove a considerable portion of bone so as to leave quite $\frac{1}{2}$-inch between the two surfaces. A piece of fascia lata, celluloid, or muscle graft should be inserted between the bony surfaces so as to ensure a false joint and prevent any bony union (Figs. 10 and 11). It is often possible to turn in a flap of the temporal muscle without extending the incision and without interfering in any way with the action of the muscle itself. In bilateral cases, after the stitches are removed, it may be necessary to move the jaw repeatedly under gas anæsthesia to ensure free movement. The end-results in these cases depend to a large extent on the patients themselves ; the persevering ones get excellent results, while the nervous patients who will not try to move their jaws often complain that the operation has been a failure and that they are little benefited by the surgical intervention. The surgeon should try to assess every patient before undertaking the operation, when he will be able to gauge the probable reaction in each case. Some patients

should never be operated upon, as failure is assured by their behaviour prior to operation.

The most difficult cases to relieve or cure are those in which there is much cicatricial contraction round about the joint. Division or excision of the adhesions is useless as, during healing, fresh adhesions form and the condition is unrelieved. In these cases, therefore, Esmarch's operation often gives good results. This operation consists in the removal of a wedge of bone, with its apex towards the alveolar border, from the neighbourhood of the angle of the mandible. A portion of the detached masseter muscle is turned in between the two bony surfaces so that an artificial joint is formed. The incision should be made below and behind the angle of the jaw ; this gives good exposure and allows the muscles to be separated from the outer and inner surfaces of the mandible. The bone can most conveniently be divided by the use of a Gigli saw. An alternate method is to remove the vertical ramus of the jaw down to the level of the alveolus, but this method does not give so satisfactory a result as Esmarch's operation.

Fig. 1
Section through mandibular joint.
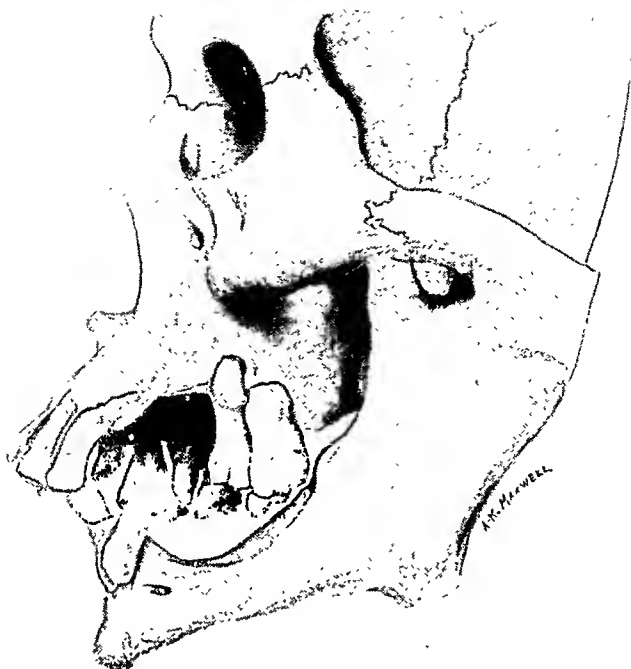

Fig. 2
Displaced mandibular disc.
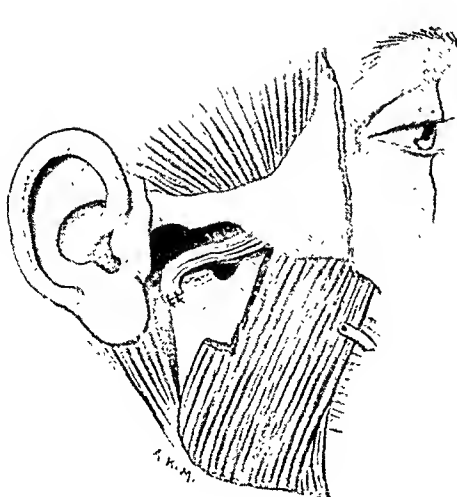
Fig. 9

Ankylosis of the mandibular joint.

Fig. 10

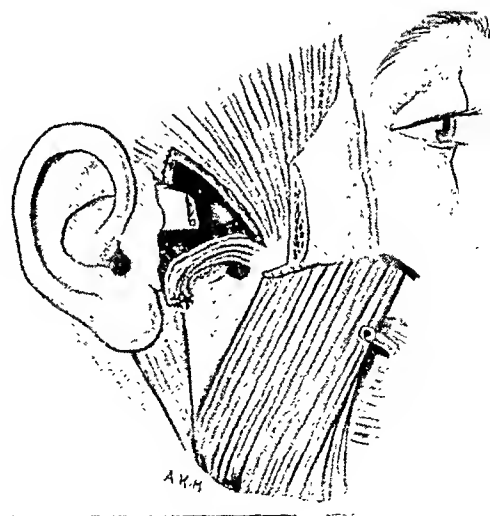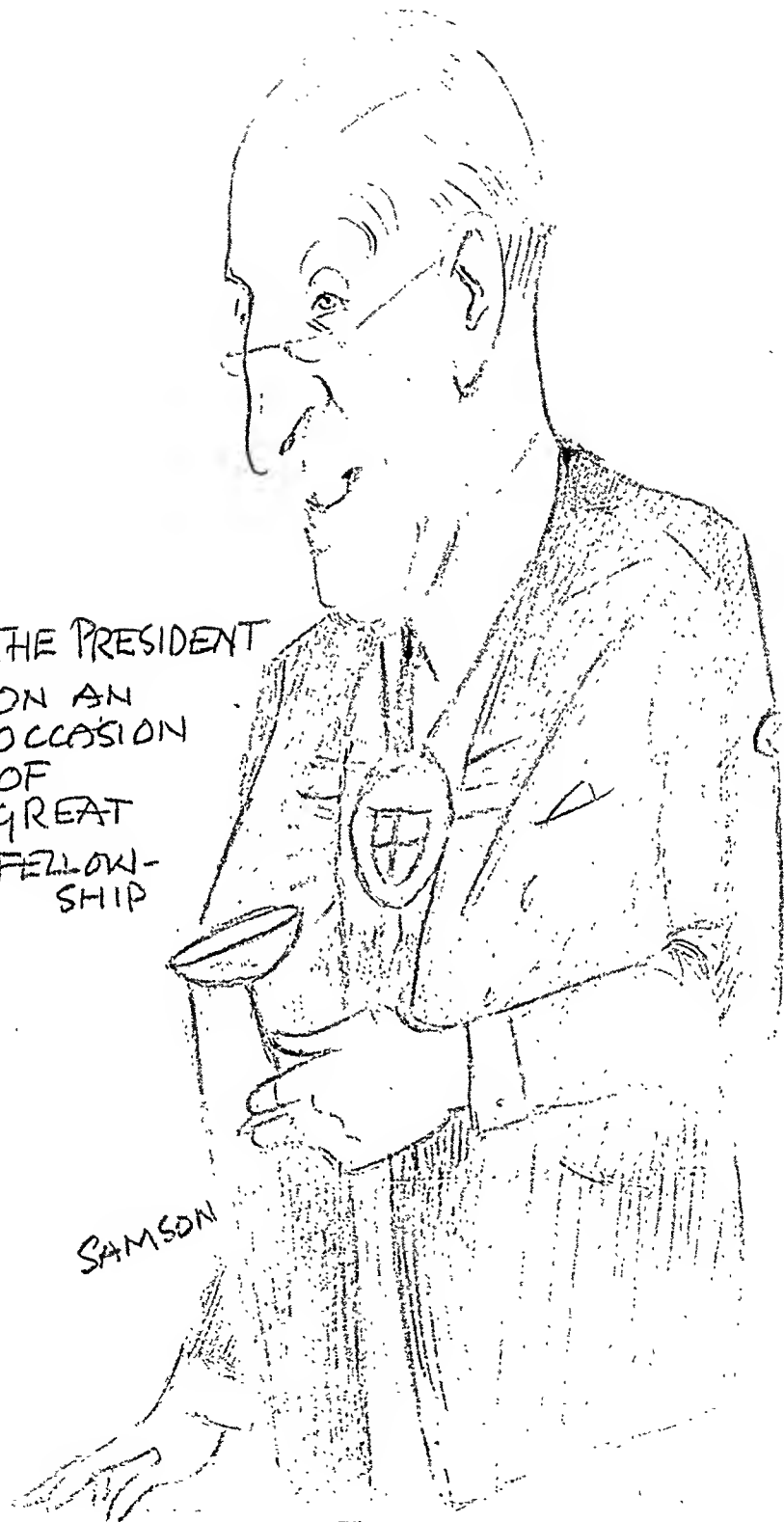Arthroplasty of mandibular joint making use of part of the masseter muscle.

Fig. 11

Arthroplasty of mandibular joint, part of the temporal muscle being used to cover the cut surface of the neck of the lower jaw.

THE PRESIDENT
ON AN
OCCASION
OF
GREAT
FELLOW-
SHIP

SAMSON

FACULTY OF DENTAL SURGERY

THE BOARD OF FACULTY held its first dinner on Friday, 30th January, 1948, when it entertained, in the Council Room of the College, some of the senior distinguished members of the Profession who had recently been elected to the Fellowship in Dental Surgery.

The President of the College (Sir Alfred Webb-Johnson) who was in the Chair, made a most inspiring speech when proposing the health of the guests. He mentioned the part that each guest had played in promoting the Art and Science of Dental Surgery. He then reviewed the history of the College and discussed its present and future policy, and stressed the important part which the new Faculty and Fellowship in Dental Surgery will play.

He took the opportunity of thanking the Board for the Silver Cup which it has presented to the College to commemorate the inauguration of the Fellowship, and the establishment of the Faculty. The cup is of George II period and bears the Arms of the Earl of Chatham (William Pitt) who was responsible for Parliament buying the John Hunter Collection and entrusting it to the College.

Mr. A. E. Rowlett in replying for the guests thanked the President and College for all they had done for Dental Surgery, particularly the establishment of the Fellowship and Faculty. They all realised how much they owed to the President who would go down in history as one of the College's greatest Presidents for his inspiration and foresight.

The Dean (Professor R. V. Bradlaw), Sir Frank Colyer and Mrs. Lindsay also spoke.

THORACO-ABDOMINAL WOUNDS IN MODERN WAR

Hunterian Lecture delivered at the Royal College of Surgeons of England
on
2nd January, 1947,
by
Guy Blackburn, M.B.E., M.Chir., F.R.C.S.,
Assistant Surgeon to Guy's Hospital

FROM TIME IMMEMORIAL the midriff and wounds of that region have interested surgeons in war. The Homeric epics, Shakespeare's writings and, of course, John Hunter's classic on " The Blood, Inflammation and Gunshot Wounds " in 1792, all refer to them—the last in detail, which is most fascinating. His observations, in fact, " of the different effects arising from the difference in the velocity of the ball " illustrate points quite applicable to the wounds of modern times. No one can gainsay his wisdom, for example, when he writes, " There may be wounds of the liver and spleen which produce no symptoms but what are immediate and may soon take on the healing disposition : but wounds in those viscera which contain extraneous matter such as the stomach, intestines, kidneys, ureters, and bladder, may produce secondary symptoms of a distinctive kind."

The experience here recorded was gained in North Africa and Italy from 1942 to 1945, and comparison with Gordon-Taylor's figures for the Great War has enabled certain conclusions to be drawn.

Mechanism of Wounding

The velocity of the missile was probably greater in the recent conflict than in 1914-18, and the complexity of modern weapons seemed more often to produce associated wounds in other parts of the body, which materially affect prognosis. Indirect wounds, due to collapse of buildings, have been excluded, but they too account for differences in large series of cases. It is still true, however, that thoraco-abdominal wounds were commoner in this war than the last, and a 26 per cent. incidence (144 thoraco-abdominal wounds in 550 abdominals) in this series—comprising cases of Estcourt, Wheeler, Rob and myself—is much higher than Gordon-Taylor's of 11-12 per cent. in the Great War and Jolly's of 11 per cent. in the Spanish Civil War. The reason would appear to relate to the methods of modern war as much as the tools with which it is waged. Italy and Africa were hilly and mountainous and direct frontal attack by infantry was costly and often unsuccessful. Flanking movements, therefore, tended to replace it and these, of necessity, produce more wounds of the flank. Coincident and steady increase in methods of attack from the air was also an important contributory factor.

Of the wounds themselves, those due to penetrating injury differed materially from those due to indirect violence, such as the " stove-in

chest " of men crushed by masonry, blast injuries of bomb and mine explosions and the under-water explosion injuries so carefully described by naval observers. The latterly-rare thoraco-abdominal accidents of parachute jumping due to early faults in harness design, described by Rene de Gaulejac and others, were likewise in a different category.

The position of the individual at the time of wounding was clearly of the utmost importance, especially in dealing with wounds of entrance only, where the missile was lodged in the tissues. Soldiers were commonly unaware of the direction from which they were injured, and localisation of foreign bodies was not always an easy matter. Shell fragments accounted for the great majority and these were not large, for large fragments, as in the abdomen, proved almost universally fatal.

Anatomy

The certainty of liver injury on the right side accounts for the better prognosis on this side as compared with the left. As an illustration, 62 right-sided wounds showed hollow viscus injury in only four and many of the patients required conservative treatment only. Of 49 personal cases, nine fell into this category without untoward result. Equally important was the great preponderance of thoraco-abdominal over abdomino-thoracic wounds (7 to 1) with a corresponding improvement in prognosis, and this has encouraged me to reiterate a decided preference, previously expressed, for the term " thoraco-abdominal " in place of the hitherto more popular " abdomino-thoracic."

The level of injury is naturally of paramount importance and damage obviously varies in expiration and respiration. Four types of projectile course have been alluded to by Hart and Seed in an analysis of 84 thoraco-abdominal wounds received at the Cook County Hospital and Cook County Morgue in 1933-37. This, after all, was modern war and it rages yet. The results have been classified as :—

    (a) The shock or hæmorrhage syndrome.
    (b) The thoracic or respiratory syndrome.
    (c) The peritoneal and retroperitoneal syndrome.

These terms are virtually self-explanatory and relate primarily to level of injury. But true gunshot wounds at close range in civil life differ from the wounds of war, where distance is measured in miles and aim is no longer a factor.

Diagnosis

This may be obvious, where, for example, herniation of lung, omentum, spleen or hollow viscera has occurred—as in the pure abdominal wound, it is not necessarily a grave prognostic sign, for it often implies a small wound of entry through which the protruded viscus or viscera cannot naturally be reduced. Such was the case in a man seen in December, 1942, with a shell wound (mortar) of the left costal margin, seen 15 hours after receipt of injury. The spleen was partly herniated and required removal through the diaphragm and the upper pole of the kidney was free

in the loin. Its removal was therefore imperative, but he made an uneventful recovery. Hæmaturia noted at catheterisation before operation in this instance had pointed to kidney damage, and this simple preliminary is a *sine qua non* of treating wounds of this type. Coincident colon and kidney damage is probably best treated by nephrectomy : simultaneous duodenal wounding on the other hand, may not need such drastic measures and the single case in this series requiring suture of a duodenal wound, cholecystectomy and conservatism with the kidney laceration (G.B.) made an uneventful recovery. Others less fortunate required nephrectomy for little more : and where gross hæmaturia lasted 48 hours or more, surgical exploration was usually deemed advisable.

The difficult cases, in respect of diagnosis, were those in which a wound of entry might or might not have passed through the diaphragm into the abdomen. Here X-rays were of the greatest value, as they were also in lesions treated conservatively. Variation in rigidity on respiration, sounds on auscultation of both chest and abdomen and the signs of diaphragmatic injury merit particular attention in clinical assessment and there may, in fact, be the equivalent of a superimposed strangulated diaphragmatic hernia. Dyspnœa, disordered heart action, shoulder pain and hiccough are associated with lesions of the diaphragmatic vault—a clinical picture described from personal experiences by Gordon Bryan in a Hunterian Lecture in 1922.

The value of X-rays, however, is even greater in the post-operative period, where aspiration of blood and air is almost invariably required and often on numerous occasions.

Associated Lesions

.Visceral wounds in combination and associated injuries of other parts of the body both affect prognosis. Porritt's analysis of thoraco-abdominal wounds in 21 Army Group in 1945 showed how favourable was single viscus injury and in a small series of 59 cases of Rob and mine the point is equally clear.

Thoraco-Abdominal Wounds
(59 cases : Mortality 46 per cent.)

	With Associated Injuries*		Without Associated Injuries	
	Alive	Dead	Alive	Dead
Left :—				
Single viscus	1	1	4	2
Multiple viscera	3	6	4	3
Right :—				
Single viscus	8	4	4	2
Multiple viscera	1	5	7	4
Total	13	16 (55·1 per cent.)	19	11 (36·6 per cent.)

*Associated injuries are only indicated when these injuries were themselves of second priority degree or greater.

Of 29 cases with associated wounds of first or second priority (major flesh wounds, amputations, open fractures of long bones, &c.), no fewer than 16 died, by comparison with 11 of 30 simple wounds.

The associated lesions took precedence at operation only where severe blood loss from them was jeopardising recovery (e.g., amputation for main vessel injury). In other cases, they were dealt with rapidly or decompressed by incision only when the thoraco-abdominal injury had already involved a time-consuming and shock-producing operation. Other errors avoided, where possible, were an inadequate appreciation of the extent of tissue damage (where the patient arrived with a reasonable blood pressure and no obvious sign of hæmorrhage) rough manipulation of liver and spleen, which restart bleeding, inadequate aspiration and excessive post-operative venoclysis. This is, in fact, a common error, for contusion of the lower lobe of the lung is common and œdema not difficult to add to it.

Treatment

Conservatism proved justifiable with right-sided lesions when injury to any viscus other than the liver seemed unlikely. The thoracic wound, however, was treated on its merits—closed if sucking, and excised if large or dirty or involving fractured ribs. Bone fragments often caused serious damage and even projected through the diaphragm, although the metallic foreign body remained on the pleural side. In two thoraco-abdominal wounds in my experience involving the heart or pericardium, bone, not metal fragment, caused the damage. Both patients fortunately survived— one a valiant little Gurkha, who treated the incident as an ignominious method of being deprived of his natural prey.

Oxygen by B.L.B. mask, the upright position, double-strength plasma and slow transfusion had their uses in pre-operative preparation and endotracheal anæsthesia, cyclopropane, positive pressure and bronchial suction were some of the anæsthetic advances that made surgery safer for these patients. Early and repeated aspiration, penicillin and X-ray control were likewise the important features of post-operative treatment or the management of cases treated without operation.

In contradistinction to the right-sided lesions, left-sided injuries as a rule presented hollow viscus damage—the small intestine, stomach and transverse colon being the commonest, with the spleen, liver and kidney in order of frequency among the solid viscera. The pancreas, from its posterior position, is naturally very uncommonly injured in cases that survive.

Methods of approach clearly differ and are guided, in a sense, by personal preference. To quote an American writer, " Thoracic surgeons are thoracically minded. General surgeons are abdominally minded. Thus they tend to approach thoraco-abdominal injuries differently." But the attitude alters with experience of and familiarity with the problems: and individual observations alone prompt me to record the following :—

1. Conservatism is wise on the right side. If operation is necessary, it should be a thoracotomy.
2. The liver prevents diaphragmatic hernia on the right side and repair is therefore not vital. Suture to the intercostal muscles is sometimes practicable in dealing with extensive tears.
3. Left-sided lesions can likewise be dealt with through the chest. Splenectomy, in fact, is easier by this than by the abdominal route.
4. An abdominal approach is necessary in the presence of a colon wound. Exteriorisation of the bowel is only practicable by this route.
5. Suction of the peritoneal cavity can only be done efficiently through an abdominal incision. Gross soiling in small or large gut wounds therefore renders this advisable.
6. Stomach lesions can easily be repaired through the diaphragm.
7. A phrenic crush is unnecessary and only aggravates the insult to the diaphragm.

The type of laparotomy wound varies with individual surgeons and the subcostal and paramedian were probably the most popular. The inference that an abdominal incision suited an abdomino-thoracic wound and a thoracic one a thoraco-abdominal wound was also fairly widely appreciated. Thoraco-laparotomy, with division of the costal margin made no appeal but it is historically the prototype. It was, in fact, practised by Bland-Sutton in 1910 on a footman aged 17, who shot himself in the chest with a revolver " because the girl he loved did not requite his love." Splenectomy and suture of the stomach, with repair of the diaphragm, were carried out and the patient recovered in spite of a B.Coli empyema. The sequel was interesting for " the girl, finding the youth so ready to sacrifice himself for unrequited love, softened her heart." In his own inimitable words Bland-Sutton adds : " The pair are contented with each other now, although the accident went near to swelling the list of love stories with tragic endings."

Complications

Of 126 cases analysed with d'Abreu in 1945, approximately a third had had a thoracotomy with trans-diaphragmatic approach to the abdomen. Twenty-six splenectomies had been done and hollow viscus injury was present in only 19. The diaphragm was studied radiologically in all and only three hernias were found—one in a Jugoslav patient, who had had no forward surgery. The diaphragm, though hurriedly repaired in several instances, appeared to have held well and suture in two layers without a phrenic crush seemed to be the method of choice.

The great value of nursing patients flat for 24-48 hours after operation was gradually appreciated and measures such as intravenous feeding, continuous gastric suction and four-hourly morphia, until intestinal

movements could be heard on auscultation, went far to diminish post-operative morbidity. Avoidance of cyanosis and retention in Forward Areas until the patient was not dyspnœic at rest in bed were also good rules of after-treatment. Evacuation in stages then or by air ambulance at 800 feet or less was the policy that paid the best dividends.

Late complications have already been discussed by d'Abreu and relate to the pleura rather than the peritoneum.

LATE COMPLICATIONS OF THORACO-ABDOMINAL INJURIES
(126 Cases)

	Cases
Hæmothorax	28
Atelectasis	15
Subphrenic abscess	12 (6 with coincident empyema)
Empyema	11
Empyema with pleuro-biliary fistula	7
Lung abscess	2
Liver abscess	2

Subphrenic abscess occurred in only 12 of 126 cases and was dealt with on classical lines—the Ochsner route, with resection of the 12th rib, being used for the posterior type. In six cases a coincident empyema needed attention. Pleuro-biliary fistula with empyema occurred in seven cases but all recovered without being conspicuously ill or developing a persistent biliary fistula or diaphragmatic hernia. Foreign bodies were removed in the majority of instances but many remained in the liver without forming abscesses. Unless the wound track led easily to them, it seemed wise to leave them alone, for liver bleeding started in an effort to find them often proved very difficult to control. Impregnated cellulose (" Oxycel ") and fibrin foam were useful in such instances and dramatic results have at times been achieved.

Results

Of 49 personal cases 34 survived, representing a recovery rate of 69·5 per cent., but nine of these were treated without operation, and all recovered.

THORACO-ABDOMINAL INJURIES
(Blackburn)

Total	49 cases	
Alive	34 cases	69·5 per cent.
Dead	15 cases	30·5 per cent.
Treated Conservatively ..	9	(No deaths)
Treated by Operation ..	40	(15 deaths : 37·5 per cent. mortality)

In the whole series of 144 cases the mortality rate was very similar (34 per cent.) and approximates to that published by Gordon-Taylor in 1918 :—

THORACO-ABDOMINAL INJURIES IN WAR

	Total	Recoveries	Deaths	Mortality per cent.
Bowlby (1917)	151	74	77	51
Wallace (1918)	70	31	39	55
Gordon-Taylor (1918)	75	48	27	36
4th Army (1918)	207	138	64	33
21 Army Group (1945)	786	353	333	42
" " "	208	128	80	38
Welch" and Tuhy (1945) and Bradford, Battle and Pasachoff	165	113	52	31·5
C.M.F. (1945), Estcourt, Wheeler, Rob and Blackburn	144	95	49	34

Comparison, however, would be fallacious and almost impossible: and the only inference to be drawn is that the agents of modern war, more lethal though they seem, still defeat the surgeon only once in every three victims who reach him with thoraco-abdominal wounds. Proportionately more surgeons and less wounded men were fortunately the rule in this War and the figures for thoraco-abdominal wounds are little inferior to those of abdominal wounds alone. The rejection rate, moreover, was negligible.

My thanks, in conclusion, are due to my colleagues for their figures and especially to Lt.-Col. d'Abreu for advice and help. To Sir Gordon Gordon-Taylor I also owe a special debt. The men and anæsthetists of my Field Surgical Units, expecially Majors Tait and Scholey, merit more than words of thanks but the greatest tribute of all is owing to the patients, who endured the vicissitudes of surgery with a fortitude and good humour that proved invincible—that, in fact, won them the long and weary struggle. As Guthrie wrote in 1848, " they are the blood, the bone, the sinew of the nation, on whose indomitable valour alone can dependence be placed in the hour of danger. By them the victory must be won, by them the loss must be sustained : and a country grateful for their services should watch over them in their necessity as a mother over her children."

BILHARZIASIS AND BILHARZIAL CANCER OF THE BLADDER

· · Hunterian Lecture delivered at the Royal College of Surgeons of England

on

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· by ·

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BILHARZIASIS is the most prevalent disease in Egypt. Practically all people engaged in farming, whether men, women or children, are infected. Bilharziasis of the bladder is usually caused by the Bilharzia Hæmatobium worm. It leads to hæmaturia and may end fatally in various ways.

HISTORICAL

· The disease is as ancient as Egypt itself. Hæmaturia was described in the papyri of Ancient Egypt and was called the A-aa disease in the ancient Egyptian language. The bladder and rectum where the ova are usually found were always removed during the process of mummyfying the bodies. In spite of that, Ruffer discovered Bilharzia ova in the kidneys of three Egyptian mummies dating 1250-1000 years B.C., i.e., more than 3,000 years old. This is the earliest authentic record of Bilharzia infection. Bilharziasis, however, was not as common then as it is to-day because at the time of the Ancient Egyptians the basin system of irrigation was employed. The Nile, during its maximum rise in August and September, was allowed to flood the land from East to West. Villages were converted into islands. The water was allowed to drain back into the Nile as it came down at the end of October. The land was cultivated merely by throwing the seeds over the surface and passing a raft to mix the seeds with the soil. After the harvest in April, the land was left baking under the burning sun of May, June and July, with temperatures of 116° or higher. This killed any Bilharzia-conveying snails and consequently Bilharziasis could only exist in lands allotted to the cultivation of fruits and vegetables, which were watered all the year round by special contrivances. That is why, in Ancient Egypt, the disease was known to affect gardeners.

Hæmaturia was also described by the Arab physicians of Medieval Egypt. From their descriptions it was apparent that hæmaturia was more common than could be explained by such conditions as a stone or tumour. In an old Arabic book of geography, written by Ibn Battouta, who visited Egypt seven centuries ago, describing the marshes of Lower Egypt, he reported that the male inhabitants of these districts used to menstruate like women. This was certainly a misinterpretation of

Bilharzial hæmaturia. Renault, in 1806, wrote a paper on hæmaturia amongst the population of Upper Egypt and Nubia. Larrey, who was Napoleon's surgeon, recorded in 1812 that hæmaturia was an endemic disease in Egypt and reported that many French troops suffered from the disease during the Egyptian campaign. Again in 1846 Pruner recorded endemic hæmaturia in Egypt.

So far hæmaturia was well known as a disease but its cause was unknown until 1851 when Theodore Bilharz, then Assistant Professor of Medicine in the School of Medicine of Cairo, discovered the causative worm in a bladder lesion. He also demonstrated ova both with lateral and terminal spines. In 1893 Manson suggested that there were two different species of Bilharzia according to differences in the anatomy of the worms, their distribution and the shape of their ova. This was verified in 1907, when Sambon established the presence of Bilharzia Mansoni as a separate species. The discovery of the first life cycle of the worm was made by Suzuki and Myiari in Japan on the Bilharzia Japonicum in 1909. They proved that the snail was an intermediate host contrary to the current belief that Bilharzia was directly transmissible from man to man. Leiper established the life history of Bilharzia hæmatobium and Bilharzia Mansoni in Egypt. McDonagh, in his book on the biology of venereal diseases, mentioned the curative effect of tartar emetic. It was Christopherson, in 1918, who proved that tartar emetic was curative. Khalil and Peter introduced another antimony compound (Fouadin) in 1929.

Ferguson, Professor of Pathology at Cairo, established the relationship between Bilharzia and carcinoma of the bladder.

The pathology, symptomatology and surgical treatment of Bilharzia were further elaborated and worked out in great detail by the classical work of Ali Ibrahim Pasha and by Professors Sorour and Makar.

It is worthy of note, however, that Bilharzia is more prevalent now than it was in Ancient and Medieval Egypt. In order to get two crops a year dams and barrages were built and the modern canal and drainage system of irrigation was introduced. The land is watered all the year round and as Bilharzia flourishes where fresh water abounds it became widespread all over the country.

Life Cycle of the Worm

The patient passes the ovum in the urine. It must reach fresh water for development. It dies in dry soil. In fresh water the osmotic pressure causes the egg to hatch in 15 minutes. It does not hatch in the urine because it is too concentrated. If the urine is diluted the eggs hatch. In cases of polyuria with dilute urine the eggs hatch and miracidia are found swimming in the urine. When the egg hatches a very active ciliated miracidium comes out and swims about in the water. The life span of the miracidium is 24-48 hours. During this time it must find its intermediate host, the snail, otherwise it dies. In the case of the hæmatobium the host is the Bulinus truncatus which is a fresh water snail which lives

mostly in canals. The miracidium pierces the fleshy part of the snail and usually lodges in its liver where it develops into a sporocyst. Active division occurs in the snail's liver and after a month the sporocyst ruptures and sheds about half a million cercariæ. These are actively motile with a bifid tail and a head. The length of each cercaria is about one millimetre and is just visible to the naked eye. These in turn swim in the water and they are attracted by heat. When the patient's skin is in contact with the water, as in bathing, the cercariæ are attracted towards the warm skin and they become attached to it by means of their suckers. The tail is discarded during the process of penetration of the skin. Special glands in the head of the cercaria secrete a proteolytic substance which dissolves the skin and the whole process of penetration is aided by reduction of surface tension. The patient feels severe itching, but by the time he begins to scratch the cercaria is already deep in the skin. The cerearia continues to burrow until it reaches a venule or a lymphatic and thus it gains the systemic circulation and reaches the heart *via* the venous blood and is distributed with the arterial blood throughout the body. Only the parasites that reach the liver survive. The parasite can be experimentally demonstrated in the liver 24 hours after penetration of the skin. In the liver the parasite reaches the portal circulation and swims in the portal vein against the blood stream. In the case of the hæmatobium the parasite reaches the vesical vessels through anastomotic channels between radicles of the inferior mesenteric vein and pelvic veins. The knowledge of anatomy displayed by the parasite is really remarkable. The parasite develops into the adult worm and lives inside small venules in the sub-mucosa and wall of the bladder. Each male worm finds a female worm. The male being a flat worm is curled longitudinally to form a canal known as the gyæcophoric canal where it bears the female all the time. The female leaves the male only temporarily to lay the ova in a still smaller and more peripheral venule in the submucosa, the idea being to lay the egg as near the bladder cavity as possible. The female worm is a rounded worm with a much smaller circumference than the male and thus it gets into smaller vessels. As the female is laying her eggs the male blocks the outlet of the venule to stop the current of blood. The eggs are laid as the female is retracting backwards towards the male. The ova penetrate the mucosa by the combined action of a tissue dissolving substance that the contained miracidium secretes and also by the squeezing action produced during the contraction of the bladder. The spine of the ovum also helps in burrowing the way through the mucous membrane. In this way the ovum reaches the bladder cavity and passes with the urine to gain fresh water again and thus the life cycle is repeated.

Although it is the Bilharzia hæmatobium that attacks the bladder yet in about 10 per cent. of the cases Bilharzia Mansoni ova with lateral spines are found in the bladder.

Once the ovum is laid it excites a reaction in the tissues around it and this is the starting point of all the pathological lesions produced by

Bilharzia. It is only the living ovum that excites a reaction. The dead ovum or the worm itself causes no reaction. Professor Sorour proved this by the intravenous injection in rabbits of suspensions of living Bilharzia ova. Bilharzial nodules were found in the lungs five days later. Another batch of rabbits was injected with a suspension heated to 55°C. for 10 minutes to kill the miracidium. No nodules were produced. The miracidium secretes a toxin the aim of which is to create tissue injury around the ovum to facilitate its expulsion into the urine. This bio-chemical activity of the miracidium is the cause of extensive bladder, ureteric, renal and other lesions. It is also the cause of cancer of the bladder which is by far the commonest form of cancer in Egypt.

In the years 1942-1946 there were the following admissions of cancers at Kasr-el-Aini and Fouad 1st Hospitals in Cairo :—

Cancer of the breast	..	..	..	378 cases	
Cancer of the rectum	..	..	..	178 „	
Cancer of the stomach		..	..	74 „	Total 967
Cancer of the cervix uteri		..	..	337 „	
Cancer of the bladder		..	..	1,022 „	

It is seen that cancer of the bladder is more common than cancer of the breast, rectum, stomach and cervix combined.

To go into the details of all Bilharzial lesions of the bladder is outside the scope of this lecture, but before dealing with Bilharzial cancer I will deal briefly with some Bilharzial lesions of the bladder, namely, hæmorrhagic areas, sandy patches, Bilharzial papilloma and Bilharzial ulcer.

When the ova are laid superficially in the submucosa of the bladder, a diffuse cellular and fairly vascular granulation tissue forms. The cells consist of plasma cells, some fibroblasts and in recent cases no eosinophils. The epithelial covering gets thickened and hypertrophied. Sooner or later the surface epithelium becomes fasciculated and loosened, breaks down and ova and blood escape. This is the main cause of hæmaturia and the presence of hæmorrhagic areas in the mucosa. If such a lesion happens to be less vascular, no fasciculation or breaking down of the epithelium takes place. Eosinophils begin to accumulate in the cellular granulation tissue and fibrosis occurs, protected by the intact thickened epithelial covering. Massive death of ova accompanied by their calcifica-tion and fibrosis in the tissues around leads to the formation of a sandy patch. These sandy patches look and feel exactly as, if grains of sand were embedded in the mucosa. The even distribution of ova over a fairly wide area leads to a plateau-like lesion. If Bilharzia ova are deposited in greater concentration in one or more foci of the lesion, the excessive local irritation occurring here produces a disturbance of tissue balance and a papilloma is produced. Consequently, flat and papillomatous lesions occur side by side. The papilloma continues to grow as long as there is

irritation. At first it is reddish and vascular but after some time the cellular granulation tissue of the core becomes gradually organized into fibrous tissue and the papilloma is blanched. Later actual calcification of the papilloma may occur. Most of the papillomata are somewhat sessile and they contain Bilharzia ova at their base and in the core. Some of them are villous and soft and they contain a lighter deposition of ova at their base. Excessive vascularity may break down a part of the papilloma leading to profuse hæmaturia and infection. The reverse, however, can occur, sloughing due to interference with the blood supply. This is how most Bilharzial ulcers occur. Pathologically, the ulcer is a manifestation of gangrene occurring in a papilloma. The deficiency of the blood supply is responsible. This in turn is due to the effect of the Bilharzial toxin secreted by the ova at the base of the papilloma. The toxin leads after some time to endarteritis obliterans, not unlike that occurring in syphilis. Superficial infection of the papilloma also plays a role. Ulceration begins on one side of the papilloma and continues until the papilloma is destroyed. The ulcer left may be rounded or irregular, but is usually elongated, its long axis being parallel to the vessels in the area. It may happen that by the time the ulcer is seen, the original papilloma has completely disappeared. Both papillomata and ulcers occur usually in the trigone of the bladder and around the ureteric orifices. Secondary infection and cystitis are a regular accompaniment. Secondary phosphatic stones may form. Excessive calcification and fibrosis in the bladder wall greatly decreases the capacity of the bladder.

Signs of severe bladder irritation and infection accompanied by hæmaturia form the clinical picture of these conditions.

The papillomata and ulcers are not pre-cancerous in the true sense of the word but they mark a stage when a carcinoma is likely to occur. They should be treated, however, because they maintain infection, irritation of the bladder, and hæmaturia.

Tartar emetic has no effect on an already existing papilloma or ulcer, but as it kills the mother worms it prevents further deposition of ova in the bladder and prevents the development of new lesions. The Bilharzial ulcer is very superficial coagulation using the unipolar tube cautery fulguration. This is followed by local and general chemotherapy and the bladder wash-outs, using increasing concentrations of silver nitrate. The immediate prognosis is good but the tendency to recurrence is very great. Either the same ulcer recurs or new ulcers appear and the treatment has to be repeated. Sessile bleeding papillomata are coagulated through a cystoscope. If large, open fulguration is the only method to be followed. Partial cystectomy is sometimes performed. If small and avascular the papillomata can be left alone. The pedunculated papillomata are easily removed by trans-urethral fulguration. If multiple, many

sittings are required. If large they can be snared after coagulating their basis.

The Relation of Cancer to Bilharzia

There is little doubt that Bilharzia is a very potent cause of bladder cancer. The points in favour of this statement are : (1) It is the most common cancer among the class commonly infected with Bilharzia. (2) The geographical distribution of cancer is more or less the same as Bilharziasis. (3) A history of Bilharzia is almost always positive. The average duration of Bilharzial infection before cancer is about 12 years. Thirty per cent. of the patients had more than one course of tartar emetic. I came across two patients with an extremely short history, being two years and 18 months respectively, and both were around 30 years of age. This is the shortest history I encountered. Apart from the history other tests for Bilharzia such as urine analysis and cystoscopy are positive. (4) Cancer cells and Bilharzia ova are associated in a very intimate manner. This shows that most cancers start in an area that was the site of ova deposition.

The Mode of Production of Cancer

The actual cause of the carcinoma is the tissue imbalance resulting from the toxin secreted by the miracidium. This toxin has well marked effects. It leads to thickening and heaping up of the epithelium, to fibrosis beneath it and to changes in the blood supply of the mucosa. The combination of these factors in other epithelial tissues in the body is known to lead to cancer, *e.g.*, lupus carcinoma, irradiation cancer, and chronic superficial glossitis. Professor Makar pointed out that calcification played an important role in the production of cancer, being found in 84 per cent. of cancers of the bladder as revealed by X-rays. He also pointed out that Bilharzial carcinoma is rare where Bilharzial calcification is rare. The bladder is the organ where Bilharzial calcification is commonly found. The role played by calcium deposits is probably this. Calcification occurs first in the mucosa; this combined with the fibrosis prevents the ova deposited deeper to it from reaching the bladder cavity. A vicious circle is thus produced. The imprisoned ova produce great quantities of toxin and this reaches a level that leads to cancer. It is worthy of note that Bilharzial infection of the female bladder is less severe than in the male and thus Bilharzial cancer is much less common in the female.

Diamantis introduced the mechanical irritation theory. He believed that during contraction of the Bilharzial bladder the basal layer of the mucosa rubbed against the calcified Bilharzia ova at the submucosa. But the bladder is very tolerant to mechanical irritation as witness a long-standing vesical stone. A stone practically never leads to cancer.

Mooro and Dolby introduced the theory that alkalinity sepsis in Bilharzial cystitis was partly responsible, but other forms of alkaline cystitis such as those produced by bacilus proteus and other organisms

practically never lead to cancer. In few cases the urine was absolutely sterile as reported by Professor Makar.

Solard and Bodelon of Toulouse reported a Bilharzial carcinoma in a Singalese in which even at autopsy there was no sepsis and consequently it appears that the Bilharzial toxin is responsible.

Pathology and Diagnosis

Microscopically three usual types are met with, *i.e.*, the Villous, or fungating, the nodular and the infiltrating. The Villous is 10 times as common as the nodular and infiltrating. Microscopically a squamous cell carcinoma, a spheroidal cell carcinoma and adenocarcinoma may occur. The adenocarcinoma is rare and has a tendency to occur in the fundus of the bladder. These cancers differ in no way from non-Bilharzial cancer except in their greater malignancy. Investigations are carried out on the usual lines. The great majority of growths are felt per rectum, suprapubically or by bimanual examination. Cystoscopy and cystograms are done but as instrumentation of these heavily infected bladders is undesirable, my colleague, Dr. Riad Fawzy, depends on urine analysis for the diagnosis and this method has worked well up till now. Urine is centrifuged and the deposit is frozen and cut. In the 10 cases of carcinoma examined, the urine was positive for cells and in one case with a more differentiated tumour a whole cell nest was found in the deposit.

Age Incidence

Bilharzial cancer occurs at a younger age than other cancers. In two personal cases the ages were 16 and 17 respectively. Professor Makar lately reported a carcinoma in a boy aged 13. The youngest case recorded was a boy of 12, by Ali Ibrahim Pasha. In a series of 360 cases of bladder cancers admitted to the section of urology, the age incidence was as follows :—

Up to 19 years	3 cases	
From 20 to 29 years	37 ,,	
,, 30 to 39 ,,	110 ,,	⎫ 218 between
,, 40 to 49 ,,	108 ,,	⎬ 30 and 49
,, 50 to 59 ,,	84 ,,	⎭
Above 60 years	18 ,,	

This shows that the highest incidence is between 30 and 39 years and that the main bulk of the cases occurs between the ages of 30 and 49. It also shows a marked decline after the age of 50 and that growths occurring below 30 years of age are twice as common as those occurring after 60. In a series of 130 cases reported by Professor Makar in 1938, 57 patients were between 30 and 40 years and another 50 patients were between 40 and 50. This again emphasises the younger age incidence.

Sex Incidence

Out of the 1,022 cases of carcinoma of the bladder admitted in the last five years 816 were males and 206 were females. This shows that

carcinoma of the bladder is four times commoner in the male. In the male it is the commonest form of cancer. In the female it comes directly next to growths of the breast and cervix but it is still four times commoner than carcinomata of the stomach and rectum combined.

Treatment

The ideal treatment is the complete extirpation of the growth. Cases suitable for this type of treatment are, unfortunately, very rare. By the time the patient seeks advice the growth is already inoperable. I know only of eight total cystectomies and 24 partial cystectomies performed. This gives a very low operability rate indeed, being about 3 per cent. This sad fact is due to various reasons :—

(1) Patients are already suffering from Bilharzial cystitis for years prior to operation. This cystitis is subject to variation and the onset of the carcinoma is regarded by the patient as a flare up of the cystitis and it is only when the condition becomes really bad that he seeks advice.

(2) The patient has suffered for years before from urinary Bilharziasis. In most of the cases the ureters are heavily infiltrated and dilated and the function of the kidneys is impaired. This in itself will make the patient a very bad subject for a radical operation. Patients are usually on the verge of uræmia and a high blood urea. Infection of the bladder is very heavy and in the presence of dilated ureters and diseased kidneys, the risk of ascending infection after operation is a very real one. To illustrate the desperate condition of the patients as we receive them the following fact should be considered. Out of 1,022 patients admitted, 157 died in hospital, giving a hospital mortality of 15·6 per cent.

(3) Bilharzial cancer is much more malignant than the non-Bilharzial one. The growths are almost always typical corresponding to Broder's Grade 3 or 4. The tendency to occur at a younger age has already been referred to. Another feature of malignancy is the tendency to widespread secondary deposits. This year in Professor Mooro's Section, I came across three cases with secondaries in bones and these were in a rib, clavicle, sternum, lumbar spine, sacrum and pelvic bones. Professor Makar has recently reported three cases with secondaries in bones, again more or less with the same distribution, i.e., the clavicle and pelvic girdle. Massive secondaries in the pelvic glands and inguinal glands are often seen. Widespread malignant infiltration of the skin of the abdominal wall, with skin nodules, and also of the thighs and groin are seen.

In addition to the usual contra-indications to a radical operation such as a poor general condition, the presence of glands, and hydro-ureters, we consider implication of the peritoneum, even without adhesion to intestines as a definite contra-indication. It is a condition too commonly found on exploration and experience has shown that recurrence is inevitable. I would add that perforation of the bladder carcinoma and general peritonitis is a frequent complication. It proved to be more

Fig. 1

A male Bilharzia worm curled inside a venule in the core of a papilloma.
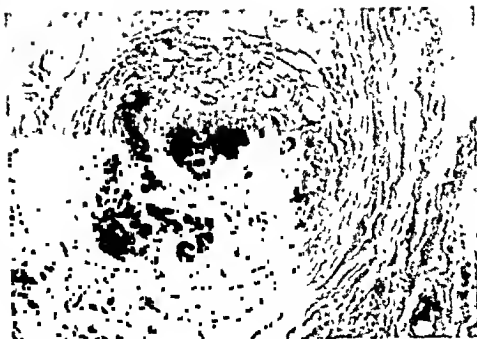

Fig. 2

A cellular Bilharzial nodule made up of epitheloid cells and Bilharzia ova.

Fig. 3

A vascular and cellular Bilharzial granulation tissue forming the core of a recent Bilharzial papilloma. Surface epithelium destroyed.
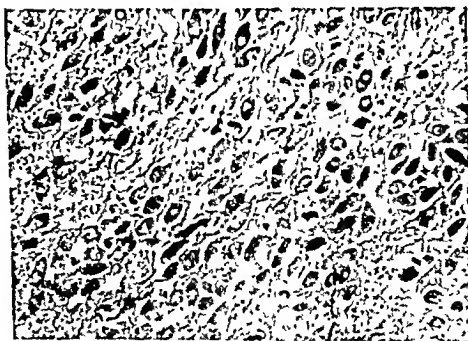

Fig. 4

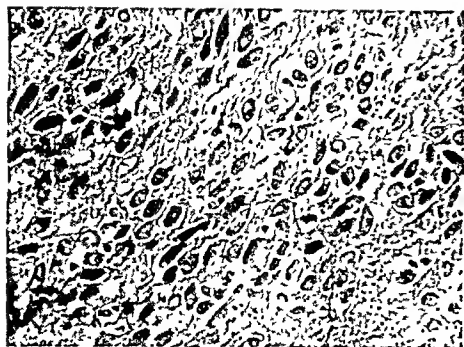A sheat of heavily calcified Bilharzia ova and fibrous tissue in submucosa of bladder.

Fig. 5

A sheat of heavily calcified Bilharzia ova in the submucosa of the bladder.

Fig. 6

Secondary in the kidney from same case showing a hæmorrhagic area in the tumour.

Fig. 7

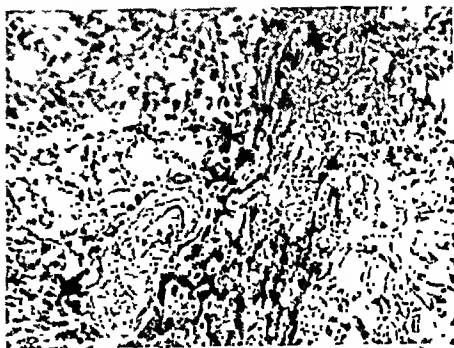Scirrhus carcinoma of bladder invading muscle with Bilharzia ova among tumour cells.

Fig. 8

Secondary carcinoma in a hæmangie-static liver from Bilharzial bladder carcinoma.

frequent than vesico-vaginal or recto-vesical fistulæ together, the ratio being 4 to 3 (Makar). All my colleagues have encountered similar cases.

The Operation of Total Cystectomy

I collected the records of six cases out of the eight performed in the last seven years. The operation is begun by a two-stage ureteric transplantation. In one case with a dilated ureter on one side, skin transplantation was performed on the dilated side and a sigmoid transplantation on the other. Three of the six died, giving a mortality of 50 per cent. One patient died six hours after the operation from shock and the two other deaths were due to uræmia. Two of the three patients who lived could not be followed, but the third was seen four and a half months after the operation and he had already massive recurrence in the pelvis.

Partial Cystectomy

Cases amenable to this operation are again few because of the reasons already stated and because in 90 per cent. of the cases the base of the bladder is either the starting place of the carcinoma or is encroached upon by the tumour. Fifteen years ago the operation was followed by radium implantation. Later the radium bomb was used. Now the operation is followed by high voltage treatment. Out of the 24 patients operated on six died, giving a mortality of 25 per cent. The main cause of death was uræmia and ascending infection. It is really unfortunate that the follow up of these hospital cases is almost impossible. Most of the patients failed to turn up again. Two patients, however, were known to be living and free from recurrence two years and three years respectively after the operation. In both these cases the growth was in the fundus of the bladder, otherwise all those who came back had recurrence.

Another point about this operation is that the bladder cavity is already greatly diminished because of Bilharzial fibrosis.

In private practice it is easier to follow up patients and I know of only two cases that could be considered as cured in a series of 40 cases treated by extensive open fulguration of the tumour followed by radium implantation or distant irradiation. One of these, a General in the Egyptian Army, developed a stone of the bladder four years after fulguration of the growth. The stone was removed and the operation of lithotomy afforded an opportunity of examining the bladder. His bladder was almost bilocular from the excessive fibrosis following radium but it was quite free from recurrence. He developed another stone two years later which was also removed. Ultimately he died nine years after the first operation from heart failure. The other case, a successful farmer, is well and alive to-day, five years after the operation. Practically all the other patients died from recurrence and consequently the rate of cure was only five per cent.

From this sad picture one can see that the great majority of patients with carcinoma of the bladder will need palliative treatment from the start. Palliative measures were tried extensively. The various methods employed were extensive fulguration of the growth, pudendal neurectomy, presacral neurectomy, combined presacral and pudendal neurectomy, ureteric transplantation, suprapubic cystostomy, intrathecal alcohol injection, stilbœsterol and the various forms of irradiation. Extensive open fulguration, or curetting out the main fungating mass of the tumour, followed by widespread coagulation of the base, both these manœuvres were found to diminish the pain and bleeding, but this relief was very short and did not last for more than three months. The method cannot be applied when the growth is extensive or when it is near the ureteric orifice. The bladder being very septic, ascending infection was very common. This line of treatment has now been abandoned. All the fulgurations were done through a suprapubic operation. Implantation of cancer in the wound is not as common as one would expect. In more than forty cases treated in this way Professor Abdalla Ali has met with one case of implantation.

Presacral Neurectomy

This is the most widely practised operation. In a total of 214 presacral neurectomies one fact emerged, namely, that this operation does relieve the suprapubic pain. Another feature of these operations is that the relief of pain is of short duration. When the patient is seen on the third or fourth day after the operation, he seems to be happier, and the suprapubic pain has vanished. When visited ten days later in at least fifty per cent. of the cases the patient states that he is beginning to get suprapubic pain again, although the pain is much milder than prior to operation. Unfortunately, as I said before, the system of " follow up " is deficient and we could only follow few cases after their discharge. From the few we saw the average period of relief seemed to vary from two to three months. Occasionally one meets with better results. I had a patient absolutely free from pain for five months after the operation and by that time the growth was felt half-way up to his umbilicus. The third fact about the operation is that in spite of the relief from pain the patient is not much happier after a presacral neurectomy. Pain is only one symptom of the disease. At the same time the patient is complaining of severe scalding during micturition, strangury, frequency, passage of pus, blood, shreds of necrotic tissue and mucus. He is debilitated from urinary infection and uræmia. These symptoms are much more troublesome than the pain. Except for frequency, the operation has practically no effect on all these symptoms. The table prepared from these 214 cases of presacral neurectomies gives the following results based on the patient's condition 15 days after the operation.

	Cases	Per Cent.	
Marked improvement	37	16·8	
Of these there was complete relief of most symptoms in	8	3·7	
Slight improvement of pain	157	73·8	90 per cent. pain relieved or improved.
Relief of perineal pain	6	2·8	
Improvement of pain and frequency	48	22·3	
Improvement of pain and dysuria	6	2·7	
Burning pain improved	10	4·6	
Marked improvement of frequency but only slight improvement of pain	2	0·9	
Mortality	8	3·7	
Burst abdomen	8	3·7	
Incontinence after operation	8	3·7	
No improvement	12	5·5	
Condition became worse	8	3·7	
Retention of urine	0	0·0	

The main cause of death was uræmia and rupture of the wound. Although the operation should increase the frequency in a normal bladder yet in a painful cancerous bladder it diminishes the frequency.

Pudendal Neurectomy (Makar)

The aim of this operation is to paralyse the external sphincter. The tone of the external sphincter is regularly increased in cases of carcinoma and the excessive contraction of the sphincter leads to strangury. The two pudendal nerves are cut distal to their branches to the sphincter ani. It is a simple and practically a subcutaneous operation and is reserved only for the worst cases that cannot stand a presacral neurectomy because of uræmia or heart failure. It is also chosen when the patient is suffering from strangury. It can be deliberately combined with a presacral neurectomy. In 17 cases treated in this way the results were as follows :—

	Cases	Per Cent.	
Slight improvement of pain	7	41·2	58·9 per cent. improved pain
Improvement of pain but very slight improvement of frequency	3	17·7	
Less frequency but very slight effect on pain ..	1	5·9	
Mortality (3 Uræmia and 1 Hæmoptysis) ..	4	23·5	
Operation	3	17·7	
No improvement	6	35·4	

The rate of incontinence after the operation is not surprising because usually the internal sphincter fails to act in advanced cases of carcinoma. The other patients did not become incontinent either because of a functioning internal sphincter, or the pudendal nerve might have branched proximal to the site of section and hence a branch was left intact, or the external sphincter might receive nerve fibres from other sacral nerves. On the whole the results of a pudendal neurectomy are inferior to a presacral neurectomy. Slight improvement occurred only in 58 per cent. of the cases, compared with 90 per cent. in the case of the presacral.

Alcohol Injection

This was again tried on a large scale (Makar). From 0·75 to 1 cc. of absolute alcohol is given either between the third and fourth, or fourth

and fifth, lumbar vertebræ, with the patient in the prone Trendelenberg's position. As a routine, Eserine tablets and one or two acetylcholine injections are given in the first 24 hours to overcome the tendency to retention. In a series of 47 cases treated in this way the results were as follows :—

	Cases	Per Cent.
Marked Improvement	6	12·8
Improvement in frequency	9	19·15
Improvement in pain	20	42·6
Improvement in Dysuria	7	14·9
The three above symptoms improved	2	4·2
Pain and frequency improved	4	8·5
Pain and Dysuria improved	4	8·5
Improved but not pain	2	4·2
Mortality (one developed paraplegia)	2	4·2
Patient got worse	1	2·1
No improvement	14	29·8
Severe headache	10	21·3
Incontinence	1	2·1
Retention and constipation	5	10·58
Weakness in lower limbs	2	4·2

Headache occurred in practically every case, but severe headache occurred in ten cases. Hypertonic glucose is given intravenously to prevent the occurrence of headache. There was a definite tendency to retention of urine but this was avoided by giving some of the choline derivatives. The patients chosen for this treatment were those considered to be bad risks for operation and those suffering from extra vesical pain. In a case accompanied by troublesome pruritus vulvæ there was complete relief from the pruritus too.

Transplantation of Ureters

It is evident that to get more relief in an inoperable case the bladder must cease as a functioning organ. This is achieved by transplantation of the ureters. The scope of this operation is very limited because of the same reasons that prevent radical treatment. Dilatation of the ureters and damaged kidneys are the rule and not exception. The three first transplantations into the sigmoid for cancer died. Cases were more carefully selected later and I collected 12 cases of transplantation with a mortality of four. Of these, one patient had only one ureter transplanted but as he developed uræmia after the first stage the other ureter was left undisturbed. Cutaneous transplantation in the case of dilated ureters is safer than transplantation into the sigmoid, but as most of the patients belong to the poor farmer class and cannot get the necessary appliance, the operation will certainly be a source of misery. The relief given after the operation is not as sensational as one would expect. The bladder is heavily infected and a discharge continues and frequent bladder wash-outs are necessary.

Transplantation of ureters, however, can be easily combined with a presacral neurectomy. This can be done when transplanting the first ureter. Slight widening of the incision in the peritoneum on the posterior

abdominal wall is all that is necessary. This combination is a very sound one and should be done as a routine when transplanting the ureters for an inoperable carcinoma of the bladder.

Irradiation

In the last five years 99 patients with carcinoma of the bladder were treated in the Radiological Department of Fouad 1st Hospital. Radium implantation has been given up. It led to flaring up of the infection, ascending infection, uræmia and a high mortality and morbidity rate. Now either the 5 gramme Radium bomb or the high voltage 400 kilo volt Maximar apparatus is used. Three anterior fields, one perineal and one sacral are used as a routine. A dose of 5,000 R is aimed at if the patient can stand it. Treatment had to be discontinued in 30 per cent. of the patients. The mortality during treatment was unexpectedly high. Ten patients died, thus giving a mortality of 10 per cent. The main cause was uræmia. It is difficult to follow up the cases but all our radiologists are agreed on the consistently poor results obtained by irradiation. A cure following irradiation alone is unknown. The few cures that I have referred to, followed actual excision of the tumour as the first step. The pain is not affected in the majority of the cases and the growth is definitely radio resistent. Some patients get worse. There is one interesting fact to note, however, in a case operated on by Professor Abdalla Ali implantation occurred in the skin around the suprapubic wound. Although the original growth in the bladder was insensitive, yet the growths in the skin were just as sensitive to irradiation as an ordinary epithelioma of the skin. I do not know if this applies to all cases of skin implantation.

Suprapubic cystostomy by itself is extremely disappointing as a palliative measure.

Stilbœsterol is being tried now at Farouk University, Alexandria, but I do not yet know of its results.

In reviewing the whole situation one finds that the picture is a very sad one. The operability rate is exceedingly low, not more than three per cent. In more than 1,000 cases of carcinoma of the bladder one can count the cures on the fingers of a single hand and for the remainder there is no really effective palliative measure. A quarter of a century ago huge chains of ureteric stones, each the size of a walnut, mammoth sized stones of the bladder and kidney, watering can perineums, penile fistulæ and cutaneous Bilharziasis were commonly seen. Now these are rare. The ravages of Bilharzia were abated as a result of great efforts and millions of pounds spent; but we still meet with cancer: As I said, the whole essence is to eradicate Bilharzia, and when Bilharzia is dead, cancer of the bladder will be an uncommon disease in Egypt.

THE BLOOD SUPPLY OF THE SYNOVIAL MEMBRANE AND INTRA-ARTICULAR STRUCTURES

by

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THE FIRST DESCRIPTION of the blood vascular pattern in the synovial membrane is that of William Hunter (1743) who described the 'circulus articuli vasculosus'. He stated: " All round the neck of the bone there is a greater number of arteries and veins which ramify into smaller branches, and communicate with one another by frequent anastomoses like those of the mesentery. This might be called the circulus articuli vasculosus, the vascular border of the joint."

Little has been said of the vascular pattern in regions of the synovial membrane removed from the articular margin, though much attention has been devoted to the superficial position of its terminal vessels following the assertion of Heuter (1866) that the capillary plexus of the synovial membrane laid naked at the synovial surfaces. That the vessels lie close to the surface cannot be denied and, as previously pointed out by Davies (1946), is a fact of considerable importance in joint pathology. That they border directly on the synovial cavity cannot now be upheld. Heuter's contention has been denied by numerous authorities such as Hagen-Torn (1882), Hammar (1894), Testut (1880) and others, and it must now be accepted that these terminal capillaries are everywhere separated from the cavity by synovial cells, connective tissue, reticular fibres or by a thin layer of amorphous ground substance. However, their proximity to the synovial fluid explains the rapidity of the exchange of solutes between this and the circulating blood.

The claim advanced by Clopton Havers (1691) that the fat pads acted as mucilaginous glands has focused attention on these structures. Rainey (1846) denied any such functional role and directed attention to the synovial fringes as a more likely source of synovial mucin. He noted the peculiar convoluted arrangement of the blood vessels in the latter and claimed that the pattern here differed essentially from that in the fat pads.

Policard (1936) has indicated that the vascularity of the synovial membrane depends on its texture. Whilst in general there is an abundant supply of capillaries and a very active circulation, he states that the aponeurotic areas have a poor circulation and a paucity of capillary vessels, whilst the more cellular areas have a fine plexus of capillaries fed by numerous precapillary arterioles. These latter he considers of special importance in so far as the control of the circulation to the capillary net at the synovial surface lies within their walls. Sappey (1859) has directed

attention to the frequent anastomoses and tortuosities of the veins in the synovial membrane, whilst Testut (1880) has spoken of their voluminous nature. In most joints these are characters which are mechanically indispensable if movement is not to interfere with the circulation, whilst the anastomoses of arteries as well as veins are frequent both in the synovial membrane and outside the joint capsule. It is well known that the blood vessels to the joints are characterised by their multiplicity, as regards numbers and sources of origin, thus ensuring an uninterrupted circulation in most positions of the joint. Testut believed the synovial membrane to be less vascular than the visceral serous membranes. It is, however, considerably more vascular than the capsular and other ligaments related to the joints, whilst its healing capacities, as compared with the latter, are undoubtedly related to this enhanced vascularity. Indeed, the speed of repair of the synovial membrane must compare very favourably with that of the serous membranes. Quain's Anatomy (1878) indicates that the vessels in the vaginal synovial membrane (tendon sheaths) are less numerous than those of the synovial membrane of the joints. This might well be so, particularly in regard to the so-called visceral layer as compared with the parietal layer of the synovial membrane, as the mechanical difficulties in supplying vessels to this layer must of necessity make the circulation within it more precarious.

Toynbee (1841) speaks of the terminal loops of the circulus articuli vasculosus as dilated beyond the diameter of ordinary capillaries. Harris (1933) has stressed the independence of the blood supply of the diaphysis of the long bones with its medullary artery from that of the epiphysis with its multiple freely anastomosing vessels which it shares with the capsule, synovial membrane and other joint structures. He expresses the unity of the blood supply to the joint and its related epiphyses by the term 'circulus vasculosus articuli et epiphyseos.'

Tobler (1929) has described the menisci of the knee as being vascularised in their outer quarter only by a capillary plexus of large mesh. Henschen (1929) has spoken of these menisci as supplied from 'une voie marginale arterielle perimeniscale.' Information concerning the relative vascularity of these menisci at different ages is not available.

The fragmentary nature of the information concerning the vascularity of the joint structures and the importance of their nutrition, especially that of the avascular articular cartilage, in the etiology of so-called degenerative joint conditions calls for further investigation.

Material and Methods

A few observations have been made on the synovial membrane in cattle. Apart from this the study has been conducted on the following human material :—

1. A full-term fœtus in which the joints of both upper and lower limbs were examined following a complete injection through the main limb arteries.

2. Lower limb of a male aged 24 years removed by a mid-thigh amputation 10 days after a gunshot injury to the femoral artery. The small blood vessels, arterioles, venules and capillaries were at this stage actively dilated and engorged with blood. No injection was required, the more superficial vessels of the synovial membrane being clearly seen when the tissue was fresh and the deeper ones becoming evident in portions removed, dehydrated and cleared.

3. Lower limb of a male aged 30 years whose death was caused by a tumour of the third ventricle. A complete injection, arterial, capillary and venous was made with Raybar emulsion.

4. Lower limb of a male aged 40 years amputated above the knee for a fibrosarcoma. The veins only were injected with Raybar emulsion. The injection was made into the dorsal venous arch of the foot.

5. Lower limb of a male aged 67 years amputated for senile gangrene. A complete injection was made through the femoral artery.

6. Lower limb of a male aged 67 years amputated above the knee for diabetic gangrene ; injected through the femoral artery to show all vessels.

7. Upper limb of a male aged 60 years, cause of death not known ; injected through the axillary artery.

Except for cases 2, 3 and 4 where injection was unnecessary or was done with a diluted suspension of barium sulphate (Raybar) as used for barium meals, the injection mass was an India ink suspension, using reconstituted human blood plasma as a basis. The latter was made from the dried plasma rejected for use on human patients on account of age. This was suspended in distilled water as indicated on the bottle ; India ink (Winsor and Newton) was added to the extent of 5 or in some cases 10 per cent. and the suspension was thoroughly mixed. This suspension was found to possess many advantages over the older injection masses. It was easily and quickly prepared as required and used cold. To obtain the best injections these had to be done slowly, any attempt at hastening the actual injection invariably resulting in incompleteness. The times taken for each injection in the present series had to be reckoned in hours. The new injection mass eliminated the difficulty of maintaining the warmth of the part during these prolonged injections and the inconvenience of handling the limb immediately after amputation. With this new method the limb could be stored in the refrigerator and when convenient thawed and injected. This procedure gave most satisfactory results and was found highly preferable to any attempt at speedy injection with partial success. Following the injection the part was fixed whole by immersion. For this it was found preferable to use a fixative which gave a firm jelly with the serum, a protein precipitant, such as alcohol. During the fixation the India ink was firmly trapped in the precipitated protein, it did not ooze out or float over the preparation during subsequent treatments, nor did it detach itself and float on to the surface during the preparation of histological sections. The precipitated blood protein adhered firmly to the vessel wall, did not shrink unevenly so that the vessels always

remained filled and when sections were cut from the tissue, after imbedding in paraffin, the ink did not get smeared over the section by the knife edge. The most complete and uniform filling of the vascular bed was obtained if the reconstituted plasma was used at double its usual strength, that is with only half the volume of distilled water indicated on the bottle. It was preferable to make the injection mass immediately before use. Any large particles in it could be removed by filtration through cotton wool or a very coarse filter.paper. Protein and ink particles tended to aggregate and settle out on prolonged standing for 24 hours. These could, however, also be filtered off and the filtrate used for injection with satisfactory results.

In these preparations the glass canula was tied into the appropriate vessel, artery or vein as required and the injection mass forced in, using water pressure from the tap, a third bottle being introduced between that receiving the water and the one containing the mass to act as a trap. Relatively low pressures, equivalent to about 100 mm. mercury were used over most of the period of injection but this was raised to as much as 180 mm. mercury towards the end of the injection in most cases in order to ensure a maximal filling of the vascular bed.

After fixation numerous small blocks of tissue were removed, dehydrated, cleared and examined with a binocular microscope. Other blocks were imbedded in paraffin or celloidin, sectioned and examined after staining lightly with Biebrich's scarlet or saffranin. The thickness of the celloidin sections required in order to see the pattern of the blood vessels varied with the vascularity of the part, in the main from about 50 to 100u. The optimum thickness was determined after examining a few trial sections within this range. The best clearing agent for examining whole blocks of tissue was oil of wintergreen (methyl salicylate). This was found to be much superior to benzene, which was used for the histological preparations.

Results

In the following description the term synovial membrane will be taken to include all the tissue lining the capsule, ligaments and periosteum ; no attempt will be made to differentiate this into synovial membrane proper and subsynovial tissues as this does not assist either the description or the understanding of the functions of the blood vessels. The term " superficial " is used to denote the innermost surface of the synovial membrane, that is towards the cavity of the joint, whilst " deep " is used when referring to the opposite surface. The vascular pattern in the synovial membrane varies with its type, whether it is areolar, fibrous or fatty, as in Key's classification (1928), and also with its thickness in the areolar areas. Where these types of membrane meet and become continuous the change is gradual both in the vascular pattern and in the nature of the membrane itself.

In the looser and more areolar areas the larger vessels, both arteries and veins, run in the deeper part of the membrane, close to the capsular tissues and parallel to the synovial surface. They branch infrequently in tree-like fashion and anastomose at irregular intervals to form wide meshed plexuses of arteries and veins. From these, branches pass inwards at intervals towards the synovial surface and, after a short course, turn parallel to the surface, branch and anastomose to form a second vascular stratum or plexus of vessels, parallel to the first or outermost plexus. The meshes and diameter of the vessels in this second plexus are finer than those of the first. This plexus sends its branches towards the surface at frequent intervals. These again, after a short course, turn to run parallel to the surface, anastomose frequently and form an irregularly quadrilateral pattern of vessels, now lying very close to the synovial surface (Fig. 1). The vessels of this terminal plexus are arterioles and venules and give off or receive the terminal capillary vessels, which form a series of finely anastomosing loops immediately under the synovial lining. The deeper plexuses of vessels give off and receive many small branches to the deeper synovial tissues ; these are short and divide in tree-like fashion to form small anastomosing capillaries. In the areolar areas, however, the number of vessels in and the density of the capillary net is concentrated mainly at or close to the joint surface, this superficial portion of the synovial membrane being much more richly supplied than the underlying tissues.

As already indicated the veins follow the arteries and, relative to the latter, they are voluminous in the deeper layers of the synovial membrane, that is in the deeper plexuses. In the latter, one, and sometimes two, veins accompany each artery, but in the finer and more superficial plexus two veins generally accompany each artery and anastomose frequently across it. Valves are frequent in all the veins, even in the most superficial plexus.

There are thus in the areolar areas of the synovial membrane some two or three vascular plexuses, placed parallel to its surfaces, the exact number, however, varying with the thickness of the membrane. These plexuses intercommunicate freely, whilst the innermost supplies a rich capillary bed to the surface of the synovial membrane. The frequency of anastomoses increases and the size of the mesh decreases in these successive plexuses as the joint surface is approached. The superficial position of the innermost and finest meshed plexus, together with that of the capillaries arising from it, is further confirmed and stressed. The dimensions of the mesh in the innermost plexus vary from 0.9 mm. to 0.15 mm.

The larger vessels of the synovial membrane enter directly into the base of the fat pads and fringes bordering on the articular margin. Here they break up in tree-like fashion, which permeates the whole structure uniformly (Fig. 2). Thus, these structures differ from the areolar areas in that there does not exist a specialised capillary net at the joint surface. When the joint fringes hypertrophy in degenerative joint disease such as osteoarthritis, the capillaries extend inwards as long loops into the hypertrophied fringes, resembling those to be described later in the

circulus articuli vasculosus. When these fringes project further and come to lie between the articular surfaces they become avascular, the capillary loops being unable to extend into these areas on account of mechanical hindrances, whilst these portions of the fringes are generally sufficiently attenuated to be nourished directly from the synovial fluid. Villi are by no means as numerous in human joints as in those of animals and most observations on these have been on cattle. Generally, a central arteriole and venule courses through the length of the villus, whilst a fine capillary net is arranged just under its surface layer. The terminal portions of many villi are avascular, whilst many very attenuated hair-like villi are wholly avascular. The strands, which are also much more frequent in animals than man and course particularly between opposed surfaces of the synovial membrane in the recesses of joints, are sometimes mainly avascular but generally have a couple of small vessels, arteriole and venule, traversing through their central or axial parts, but giving off few capillaries during this part of their course. These strands are usually composed of very compact fibrous tissue.

The vascular pattern in the ligaments and capsules bears close similarity to that described by Edwards (1946) in tendons. The larger vessels, arterioles and venules, usually arranged in groups of three or more, course longitudinally between the fascicles and fibrous tissue bundles and are fed from vessels placed outside the joint. The longitudinal vessels communicate frequently by smaller vessels running transversely and around the connective tissue bundles (Fig. 3). Where the ligaments or the capsule are attached to bone the superficial vessels anastomose with those of the periosteum, whilst the deeper vessels end either as tufted loops at the bony or cartilaginous surface or, in the adult, continue into the bone to anastomose with the vessels of the epiphysis in the circulus vasculosus et epiphyseos of Harris. In the young, when the epiphyses are unossified, a few of these vessels also supply the cartilage canals, there being within most of these a central arteriole and venule, whilst on the walls of the canal is a delicate capillary network. These canals are found in all parts of the epiphysial cartilage and contain its only vessels. They were first described by Bubnoff in 1868. The main vessels here branch to continue into the various subdivisions of these canals. The few terminal loops supplying the cartilage canals are seen at the attachments of the cruciate ligaments to the proximal surface of the tibia in the newborn (Fig. 4). At the attachment of the ligamentum teres to the head of the femur it is interesting to note that Walmsley considers that the vessels coursing in the ligament play but a small role in the supply of this epiphysis, whilst he quotes Hyrtl as stating that the arterial vessel in the ligament forms a capillary loop at its femoral extremity and runs back as a vein. Similarly at the attachments of the cruciate ligaments the terminal capillary loops form by far the most conspicuous feature, whilst the number of cartilage canals supplied is small. Where the synovial membrane lining these ligaments is thin and firmly blended with them,

it is supplied directly by capillary vessels which pass inwards from the longitudinal vessels of the ligament, so that the capillaries tend to form a longitudinally arranged series of loops. Where there is a greater thickness of synovial membrane, however, it has its own independent vessels, which course tortuously over the ligament, anastomose with the longitudinal vessels on and between the fascicles and supply a capillary network to the membrane itself. The capillary bed here is not so well developed as that in the more areolar areas of the synovial membrane. The vessels of this fibrous synovial membrane end at the articular surfaces in the typical looped anastomoses of Hunter's circulus articuli vasculosus, extending a short distance on to the articular cartilage (Fig. 5). This feature is again well seen at the attachments of the cruciate ligaments to both tibia and femur.

A few ligaments require special mention. The first of these is the annular (orbicular) ligament of the proximal radio-ulnar joint. At its attachments to bone this ligament and its lining synovial membrane is supplied by a plexus resembling that seen in the superficial layers of the areolar membrane. Elsewhere, where the ligament approaches a fibro-cartilaginous consistency and the synovial membrane is firmly plastered to its surface without the intervention of loose tissue, or even blended with the underlying ligament, the blood supply is mainly from circularly running vessels placed at the upper and lower margins of the ligament. These anastomose by vessels running perpendicularly, placed at intervals, the pattern resembling the rungs of a ladder, whilst the intervening membrane is sparsely supplied by a few fine anastomosing vessels. The vascularity of the main part of the ligament is decidedly poor.

Another ligament with a distinctive vascular pattern is the plantar calcaneo-navicular or spring ligament (Fig. 12). Here, though the main vessels run in the direction of the fibres, an appreciable number of vessels enter the ligament along its medial and lateral margins. As these vessels approach the fibrocartilaginous central area or facet on which the head of the talus rests, they become attenuated and the majority end in fine capillary loops directed towards this central area. As in the annular (orbicular) ligament there is no separate supply here to the thin lining of synovial cells over most of the upper surface of the ligament. A pattern similar to that in the spring ligament is found in the accessory volar (glenoid) ligaments of the metacarpophalangeal, metatarsophalangeal and interphalangeal joints where these ligaments are fibrocartilaginous in consistency and form part of the articular surfaces for the heads of the metacarpals, metatarsals or phalanges. This pattern is also seen in the posterior parts of the knee joint capsule where the posterior aspects of the condyles impinge on this in full extension. In all these areas the ligaments mentioned form part of the articular surface and the pattern of the vessels may be considered as an imperfect formation of a circulus articuli vasculosus around the articular, in most of these places, fibro-cartilaginous areas.

Hunter's circulus articuli vasculosus occurs where the synovial membrane overlaps the articular cartilage and ends by blending with its superficial layers. As the articular areas are approached, the synovial membrane becomes thinner and the larger blood vessels, both arteries and veins, come to lie closer to the surface (Fig. 6). Here they anastomose with those of the epiphysis, the voluminous nature of the anastomosing veins being particularly evident. In the young these vessels supply the branched and unbranched cartilage canals of the cartilaginous epiphysis. These canals pervade all parts of the epiphysis at this stage but never enter the articular cartilage, their extremities in the region of the latter pointing towards and lying directly beneath the articular cartilage (Figs. 7 and 8). The larger vessels of the synovial membrane as they approach the weight-bearing areas branch repeatedly and anastomose to form a series of arcades like those of the mesentery of the small gut, so aptly described by William Hunter; they terminate in a fringe of looped anastomosing vessels over the non-weight-bearing part of the articular cartilage (Fig. 6). The length of these terminal loops varies with the width of the non-weight-bearing peripheral area of cartilage and whilst, in the human, these terminal loops are characteristically straight, and, as pointed out by Toynbee, more voluminous than ordinary capillaries, in other animals, such as the ox, these vessels are typically wavy, irregular and much finer. The circulus articuli vasculosus lies adjacent to and covers that area of articular cartilage which develops osteophytic outgrowths in degenerative joint diseases. It is undoubtedly the terminal plexus from which the peripheral part of the articular cartilage receives its nutrition.

As regards the blood supply of the intra-articular discs the superficial vessels of the synovial membrane terminate where this merges with the disc in a series of capillary loops comparable with those of the circulus articuli vasculosus. At the horns of the discs in the knee joint these vessels extend inwards for a short distance, particularly along its inner concave margin (Figs. 9 and 11). This extension inwards is more marked in the newborn than later but traces of its can be found at all ages. Between the two layers of synovial membrane attaching to the upper and lower surfaces respectively of the discs in the knee joint are larger vessels running circumferentially around the outer concave margin of the disc. These vessels run from the anterior and posterior horns but are reinforced elsewhere by the deeper vessels of the synovial membrane. They constitute the perimeniscal vessels of Policard (1936). From them vessels pass radially inwards into the disc, anastomose by short lateral branches and end in looped capillaries in the substance of the disc. (Fig. 10). The inner portion of the disc is devoid of blood vessels, this avascular portion increasing proportionately with age. Thus, in the newborn, in a medial disc of average width of 3 mm. the peripheral 1·5 mm. is vascularised, whilst in the corresponding lateral disc the average width is 4 mm., of which the peripheral 2 mm. are vascularised. On the other hand, in an

adult male of 40 years the medial semilunar disc is 13 mm. in width, of which only the peripheral 2 mm. are vascularised, whilst in the lateral semilunar disc of 14 mm. width, blood vessels penetrate into the peripheral 2·5 mm., leaving 11·5 mm. avascular. In other words, the avascular part of the disc increases both absolutely and proportionately during the growth period without any corresponding extension inward of the peripheral vessels, so that the absolute width of the vascular area of the semilunar cartilage remains approximately the same at all ages. The degree of vascularisation of the semilunar discs is maximal at the two horns but decreases here with age. In the newborn, T.P. McMurray speaks of the perimeniscal artery sending branches to the middle and inner zones and suggests that this is a free supply of vessels at this stage spreading from the outer border to the inner sharp margin. He also describes a devascularisation of the inner zone of the meniscus with age. The peripheral blood supply to the lateral semilunar disc is interrupted where this is crossed by the tendon of the popliteus, the portion of disc opposite the centre of this pattern of the vessels in the disc of the inferior radio-ulnar joint is similar to that seen in the discs of the knee joint, the blood supply coming mainly along looped capillaries from larger vessels running at its anterior and posterior margins. These capillaries extend further into the disc along its attachment to the radius, those from the front and back of the joint anastomosing along this line. The central and greater part of the disc is avascular.

Injected specimens of the disc in the temporo-mandibular joint have not been available but histological sections of uninjected specimens show that here again the central and major part of the disc is avascular, the blood vessels being confined to its peripheral parts. A noticeable feature in sections of this disc is the presence of large venous spaces close to and within its medial and posterior portions. These are presumably connected with the pterygoid plexus of veins and form irregular endothelially lined clefts in the peripheral parts of the disc. The walls of these spaces are supplied with an abundance of elastic tissue.

There is a particularly rich supply of valves even in the smaller veins of the synovial membrane. They are most numerous in the more areolar areas and are plentiful even in the innermost plexus of vessels. They are also evident in sections of the perimeniscal vessels.

Discussion

The blood supply of the synovial membrane and other intra-articular structures presents several features indicating adaptation to physiological requirements. Firstly, the multiple sources of origin of the arteries and the multiplicity and voluminous nature of the terminal veins together with the rich anastomoses of the branches or tributaries of these within the synovial membrane ensures an adequate blood supply to the joints in all the positions or attitudes which these may assume. Furthermore, these factors explain sufficiently the immunity of the joints, together with

the associated bones with which they share their blood supply, from involvement in peripheral vascular disorders until the late stages. The efficient venous drainage and the rich complement of valves in the veins adequately explain the freedom which the joints enjoy from effusion in congestive cardiac failure.

The richness and superficial position of the terminal capillary network in the areolar areas of the synovial membrane indicates that these are the more active regions in the rapid physiological exchange which exists between the blood and the synovial fluid and, which is so important in the metabolism of the large areas of avascular articular and other cartilage within the joints. Similarly, the efficiency of the blood supply to the more peripheral parts of the articular cartilage from the circulus articuli vasculosus endows these peripheral areas with reparative and proliferative properties not possessed by the more central areas which are nourished indirectly through the synovial fluid. The more uniform arrangement of the smaller blood vessels throughout the fat pads of the synovial membrane indicates a function distinct from that of the more areolar areas.

From the pathological viewpoint the superficial nature and richness of synovial capillaries accounts for the ease with which blood is extravasated into joints. As Davies has previously indicated, a few red cells in the synovial fluid are almost inevitable, following simple puncture of the joint whilst small hæmorrhages into joints occur even after relatively trivial injuries. Furthermore, the expanse of the capillary network at the synovial surface is sufficient to explain the rapidity with which serious effusions collect in joints following injury or internal derangement. The mechanism whereby such effusions are produced is by no means clear. Nor is the role of the nervous system in controlling the synovial capillaries and their permeability understood and a further investigation is required on the work of Engel (1940-41) and Engel and Forrai (1943), who found a selective permeability of the synovial membranes to acid dyes, together with an unexpected decrease in the rate of filtration from the blood into the synovial fluid following sympathectomy. It would appear also from the work of Engel, and contrary to the usual belief, that the permeability of the synovial vessels is considerably decreased as compared with the normal in inflammatory conditions of joints but the richness and superficial position of the synovial vessels provides still a large and rapid absorptive area so that the toxic absorption in infective joint conditions, particularly of the larger joints, remains a factor of the utmost importance. Further, the changed permeability of the synovial barrier in inflammation is important in determining the route of administration of therapeutic agents for its control.

The villi and fringes are undoubtedly less efficiently supplied with blood than other portions of the synovial membrane. Their vessels of supply and drainage are frequently single. Consequently, in its vascular reactions, whether resulting from inflammation or allergic causes, the

earliest changes in vaso-dilatation and engorgement are seen in these regions. The loose areolar areas of synovial membrane are the next to be affected, whilst the least vascular and the last to display any change are the more fibrous areas over the ligaments. Furthermore, the œdema and swelling resulting from the pathological process is never as marked in these areas as elsewhere in the membrane.

The small, or even negligible, role which the ligamentum teres plays in vascularisation and nourishment of the head of the femur has been stressed by Walmsley (1915). Similarly, it would appear that the vessels of the cruciate ligaments supply but a minimal part of the blood to the epiphyses at the knee, as only a few vessels pass into the cartilage canals in this region in the newborn. Indeed, a major role in the nourishment of epiphyses can hardly be expected for intra-articular ligaments, their position and vulnerability tending to make them unsuitable for such a function.

The blood supply of the semilunar discs of the knee and other intra-articular discs is interesting in regard to their repair and the occurrence of cystic degeneration within them. As regards attempts at the healing of tears in the semilunar cartilage little is known. Mechanical and vascular factors here are undoubtedly too unfavourable for any degree of repair to occur. Repair has, however, been adequately observed in these discs in dogs following experimental injury; information is still required as to its occurrence in man and the incidence and position of granulation tissue in torn discs. A systematic search for granulation tissue might supply some valuable information as to the possibilities of repair.

As regards the genesis of the cysts seen in the semilunar discs in the knee joint, particularly in the lateral meniscus, there are still two opposed views :—

1. That they are of the nature of congenital abnormalities, endothelial inclusions, trauma leading to irritation and distension of these (Ollerenshaw, 1935).

2. That they are cystic or mucoid degenerations in fibrocartilage and fibrous tissue resulting from trauma, but without any previous lesion in the disc (Allison & O'Connor, 1926 : Bristow, 1928).

The arrangement of the blood supply of the menisci would undoubtedly tend to favour the latter view. In both menisci the proportion of avascular to vascular tissue is relatively low at birth as compared with the adult. The avascular segment of the disc must of necessity derive its nourishment from two sources. At its surfaces and throughout its thickness towards its inner free margin it must be entirely nourished from the synovial fluid. In its thicker peripheral part its surface layers are nourished from the circulus articuli vasculosus which extends inwards for a distance slightly less than 2 mm., whilst its deeper layers are nourished from the terminal loops which pass inwards from the perimeniscal artery for a distance of 2 mm. or slightly more. A central wedge-shaped area extending inwards from this vascular part at the periphery probably receives an impoverished

Fig. 1

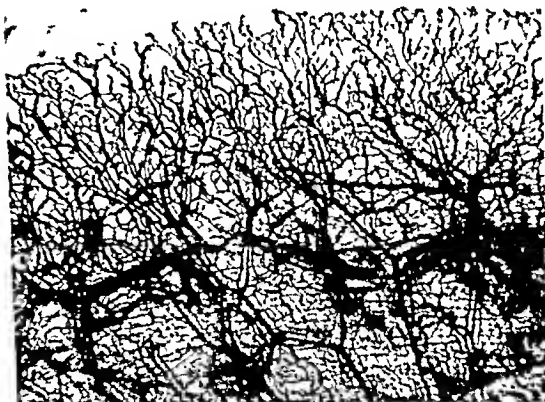Fig. 2

Fig. 3

Fig. 4

Fig. 5

Fig. 6

Fig. 1.—The blood vessels of an areolar area of synovial membrane in the knee-joint of a man aged 67 years. x 34.

Fig. 2.—The blood vessels in a portion of the infrapatellar pad of fat of a man aged 67 years. x 30.

Fig. 3.—The blood vessels in the ligamentum patellæ of a human newborn. x 30.

Fig. 4.—The blood vessels in a cartilage canal in the upper end of the tibia in a human newborn. These vessels are connected with those of the anterior cruciate ligament which can be seen in the lower left-hand corner of the photograph. x 2?.

Fig. 5.—Termination of the blood vessels of the synovial membrane covering the anterior cruciate ligament at the upper articular surface of the tibia. x 19.

Fig. 6.—The vessels of the circulus articuli vasculosus on the medial side of the medial femoral condyle in the newborn child. The darker regions within the synovial membrane, where vessels pass into or leave the articular surface of the medial femoral condyle x 27.

Fig. 7

Fig 8

Fig. 9

Fig. 10

Fig 11

Fig. 12

Fig. 13

Fig. 14

Fig. 7.—The blood vessels of the cartilage canals in the lateral femoral condyle approaching the articular cartilage but not penetrating into it Newborn child x 12.

Fig. 8.—Blood vessels in a cartilage canal in the lower femoral epiphysis of a human newborn. They are seen connecting below with the blood vessels of the synovial membrane, x 19.

Fig. 9.—Low power view of the medial semilunar disc of a newborn child to show the extensions of the blood vessels along its inner margin at the anterior and posterior horns, x 3.

Fig. 10.—High power view of the peripheral capillary loops in the medial semilunar disc shown in Fig. 9, x 15.

Fig. 11.—High power view of the extension of blood vessels along the inner margin of the medial semilunar disc shown in Fig. 9 from the posterior horn, x 30.

Fig. 12.—Blood vessels of the plantar calcaneo-navicular (spring) ligament in a human aged 67 years. The numerous terminal loops around the central area supporting the head of the talus are well shown, x 24.

Fig. 13.—Section through a portion of a cystic lateral semilunar disc of a male aged 34 years, showing granulation tissue around the blood vessels close to the cystic spaces, x 95.

Fig. 14.—Section through a portion of a cystic lateral semilunar disc to show the absence of any definite lining to the cysts in this position and the proliferation of the cartilage cells which are arranged in large groups close to the walls of the cystic spaces. From a male aged 18 years, x 245.

nutritive supply, partly from the terminal loops at the periphery, and partly from the synovial fluid. In the normal meniscus this wedge-shaped area is easily identified by the paucity of cells within it. Here the number of cells per unit area in section is minimal, increasing in all directions as one passes towards the surface of the disc. With growth the disc increases in width and height, the actively growing area being the peripheral vascular region. This growth keeps pace with that of the joint as a whole, and with that of the skeletal tissues in general, whilst there is a concomitant increase in density of the tissues of the disc, the compactness of its fibrous tissue increasing whilst the elasticity of the disc decreases as a result.

The amount of elastic tissue in the disc is small at all ages, the elasticity being mainly determined by the arrangement of the fibrous tissue bundles traversing it. A much greater amount of elastic tissue occurs in the disc of the temporo-mandibular joint than in those of the knee. All these growth changes occur without any further penetration of the disc by the blood vessels situated at the periphery, in other words the nutrition of the disc, and particularly of the subperipheral area, becomes increasingly precarious, the avascular portion is, as it were, tending to outstrip its source of nourishment. The discs of the inferior radio-ulnar, sterno-clavicular and temporo-mandibular joints remain relatively small and are thin as the supply of nourishment and volume of tissue to be supplied is by no means as great in these regions as in the knee. Furthermore, these discs enjoy an immunity from trauma not shared by the knee joint. It is interesting to note that the maximal incidence of cysts in the lateral semilunar disc is reached between 20 and 30 years of age, when they are attaining their greatest dimensions, towards the end of the growth period, when the discrepancy between the dimensions and blood supply is maximal. Trauma puts an added strain on the nutritive demands which at this age may exceed any reaction on the part of the peripheral vessels and result in mucoid degeneration. The degeneration in the disc occurs in a wedge-shaped area extending inward from its peripheral surfaces and not, except in rare cases, reaching its articular surface. These cysts may rupture secondarily into the joint cavity, possibly on account of some added trauma or injury. In some cases there is an attempt at proliferation of new capillary loops from the periphery into the devitalised and degenerating areas with the formation of tracts of granulation tissue in their wake (Fig. 13). Mechanical conditions, however, prevent these vascular reactions being sufficient to inhibit the degeneration in many cases.

The greater incidence of cystic degeneration on the lateral side is undoubtedly due in part to the greater susceptibility of this disc to direct injury. Harris (1934) has suggested that cystic degeneration is more frequent in the lateral meniscus than in the medial, as it is related to the weight-transmitting external condyle. He has also described calcification, ossification and bone marrow formation as a normal feature in the menisci of the adult rat. Cystic degeneration has also been seen frequently

in association with tears of the lateral cartilage. Subperipheral tears (bucket handle) would, in most cases, however, place the wedge-shaped area of cartilage with a precarious nutritive supply in closer contact with the synovial fluid and thus improve its nutrition. It is also frequently stated that the higher incidence of cysts in the lateral cartilage is related to its greater height. Both Quain and Piersol's (1907) Anatomy state that the lateral meniscus is deeper. The few measurements of the discs available in the literature do not bear out this statement. Thus, Fick (1904) gives the following figures for the height of the two menisci in the knee :—

Meniscus	Height		
	Anteriorly	Midpoint	Posteriorly
Medial	1·5—3·5 mm.	5·0—6·5 mm.	6·0—7·5 mm.
Lateral	4·5—5·5 mm.	5·0—5·5 mm.	5·0—6·0 mm.

A discoid semilunar cartilage is generally of greater height than the normal ; this, with its increased breadth, predisposes it to cystic degeneration, as the nutritive problem here is considerably more acute. On the other hand, Ollerenshaw (1929 and 1935) regards this congenital abnormality of the meniscus as being frequently associated with congenital inclusions and thus more liable to cystic development. How these inclusions arise in the embryo is not explained. To complete the etiological picture he has also to consider the discoid meniscus as specially predisposed to trauma, whereas if it is merely considered as a nutritive problem, trauma need not even be involved as the precipitating factor.

Finally, comparing the medial and lateral menisci, apart from liability to trauma of a special kind, that is direct trauma without tearing, there is another factor which predisposes the lateral disc to cystic degeneration, the avascular, or almost avascular, area at its periphery where it is crossed by the tendon of popliteus. This region, where the disc is grooved by the tendon of popliteus is up to 1 cm. wide, leaving a wedge of the meniscus extending inwards from this, totally devoid of blood vessels even at the periphery. Though there is no information in the literature as to the exact sites of cystic degeneration it would appear that these are probably commonest just in front of the tendon of popliteus. The cysts would tend to project outwards either at its anterior or posterior margins and data on this point are needed. Conversely, the better blood supply at the horns of the semilunar cartilages would make these regions less liable to cyst formation, whilst the medial cartilage, increasing in breadth on passing backwards would in all probability have its lowest nutritive supply at or just behind its midpoint. These views need further testing in the light of an accurate plotting of the sites at which these cysts occur.

Though not directly related to the blood supply of the discs, there remains one further point for consideration, namely, the lining of the cysts. Ollerenshaw describes it as endothelial. It is unnecessary here to digress on the definition and usage of this term, though Jenkins (quoted by Ollerenshaw) does attempt a definition in one of the latter's papers. The term undoubtedly has come to include a number of diverse tissues.

No specific staining reactions can be described for these and so its identification is in large part a matter of personal standards. However, many of the cystic spaces, both small and large, seen in degeneration of both lateral and medial semilunar discs are devoid of any special lining or cells in their walls, these latter being formed merely by the connective tissue fibres and ground substance of the meniscus. Others show a lining of flattened connective tissue cells of variable depth, obviously having undergone some proliferation. Co-existing with this, or occurring independently of it, there frequently occurs around the walls of these cystic spaces some proliferation of cartilage cells, so that groups of two, four or more cells are seen as in proliferating cartilage elsewhere (Fig. 14). These cartilage and fibrous tissue cells bordering on the cyst may occasionally become swollen and granular with pyknotic nuclei before finally degenerating and disappearing into the mucoid substance within the cyst. These features are all worthy of note, as in avascular tissue they represent a very fundamental reaction which replaces the inflammatory reaction of the vascular tissues. In the former, the reaction consists either of a reversion of the tissue to its embryonic form, namely, fibroblasts or fibrocytes, or in proliferation. Both these reactions may occur in neighbouring areas of the same tissue. They are reactions of fundamental importance in the pathology of the joint tissues.

Summary

1. A simple and convenient method of injecting the blood vascular system is described.

2. The blood supply of the synovial membrane and intra-articular structures is studied by this method.

3. The features of this blood supply of joints are described and their bearing on the physiology and pathology of the joint tissues discussed.

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"OBSERVABLES" AT THE ROYAL COLLEGE OF SURGEONS
3. THE RANBY CUP

JOHN RANBY (1703-1773) BECAME a Member of the Company of Barbers and Surgeons in 1722. He was made Surgeon to King George II's household in 1738, Sergeant-Surgeon in 1740 and Principal Sergeant-Surgeon in 1743.

Ranby accompanied George II on the German campaign and was present at the battle of Dettingen in 1743, where he earned the good graces of the King by his care of his third son, young William Augustus, Duke of Cumberland (aged 22), who had a musket-ball through his leg.

In 1745 Ranby's influence with the King and Government secured the passing of the Act of Parliament dissolving the United Company of Barbers and Surgeons and establishing two separate Companies. Ranby was the first Master of the Company of Surgeons, though he had never held office in the Barber Surgeons Company, and upon taking his seat he presented a handsome silver cup, which is still in the possession of the College. It is a standing two-handled cup with cover, and measures 15½ inches in height and 7½ inches in diameter at the rim. The hallmark shows that the cup was made in London in 1745, and it bears the maker's mark E. G. (Elizabeth Godfrey).

The inscription may be translated, "John Ranby dedicates this memorial, such as it is, to the very Worshipful Company of Surgeons on the first day of July, 1745, as a token of regard for his brethren."

Engraved on the Cup are the Arms of the Company of Surgeons—not the Arms of the College as stated by Sir D'Arcy Power in the British

The Ranby Cup, inscribed as follows:

Die July primo, MDCCXLV
Hoc Qualecunque suae in Fratres
Observantiae Monumentum
Dignissimae Chirurgorum
Londinensium Societati
Consecrat Joannes Ranby.

Journal of Surgery of October 1932. This is one of the few remaining representations of the Arms of the Company of Surgeons now existing. The Master's Chair on which the Arms were painted was destroyed when the College was bombed in 1941.

The Company of Surgeons held their first meetings in Stationers' Hall, and eventually secured premises in the Old Bailey where they built Surgeons' Hall and entered into occupation in 1751. They then elected Ranby as Master for the second time and re-elected him for the third time in 1752.

The College does not possess a portrait of Ranby and efforts to trace one elsewhere have met with no success. He was reputed to be a man of stormy passions, harsh voice and inelegant manners. Fielding introduced him into his famous " History of Tom Jones'". as " Mr. R———, Sergeant-Surgeon to the King, and having the first character in his profession."

Ranby's chief work, " The Method of Treating Gun-shot Wounds," gives an account of some of the cases he saw when serving under Lord Stair in the German campaign.

His pamphlet on the last illness of the Earl of Orford (Sir Robert Walpole) caused great offence to the physicians, for in it he utterly condemned the use of the " Lithontryptic lixivium " in the treatment of stone.

The most interesting historical episode in Ranby's surgical career was his attendance on Queen Caroline during her last illness. A complete account of her illness and death from strangulated umbilical hernia is to be found in Lord Hervey's " Memoirs of the Reign of George II," in which there are vivid satirical pictures of the Court. Hervey (1696-1743), as Vice-Chamberlain, exercised great influence over Queen Caroline. He was also a close friend of Lady Mary Wortly Montagu who introduced into England the practice of inoculation against smallpox.

Hervey's account of Queen Caroline's illness gives an illuminating impression of the state of abdominal surgery in Ranby's time. It is readily available to surgeons, as it was quoted in full in an article by Sir D'Arcy Power in the *British Journal of Surgery* (Volume 20, July and October, 1932).

<div align="right">A. W-J.</div>

THE LIBRARY
Historical Landmarks of Anatomy and Surgery

THE COUNCIL of the College, in reviewing the scope and purpose of the Library, deems it important to collect the literature of surgery and its ancillary sciences, both contemporary and historical, while avoiding the non-surgical specialisms of medicine whose literature is now more appropriately cared for by other libraries in London. Opportunity has accordingly been taken both to dispose of unsuitable books from the Library, as for instance by the transfer to the London School of Hygiene of a large collection of official public health reports, and to revive the old tradition of collecting original historical texts, a tradition established in the first great period of the College's growth a hundred years ago, when the Fellowship was founded and the Museum and Library were actively developed. At that time the foundations were laid of our very representative collection of surgical books of all periods, and it is gratifying now to record the acquisition by gift and purchase of many notable additions to this collection during recent months, after a long period when such accessions were but occasional. History is continually expanding— " Always at my back I hear Time's winged chariot hurrying near "—and it will be seen that the books recorded below cover every period of surgical history, from the re-birth of science four hundred years ago to the threshold of to-day and the work of men but lately gone from us.

" Anatomy," says Professor Charles Singer, " was the first observational discipline to be studied on modern scientific lines," and it received that distinction through the genius of Andreas Vesalius. The Library has a good collection of Vesalius' various works, which was studied by Harvey Cushing on his last visit to England, while preparing his monumental bibliography of Vesalius literature ; we have one edition apparently unknown elsewhere. The Council has lately taken the opportunity to improve our series of editions of his greatest work, the *Fabrica* or " Workshop of the human body," by buying fine copies of the third and fourth illustrated editions published at Venice in 1568 and 1604. The College already possesses the famous first edition of 1543, and one of the finest copies known of the 1555 edition, presented some years before the war in memory of Bilton Pollard, F.R.C.S. These books are not to be thought of as playthings for the bibliophile ; they are essential documents in the story of medicine, and the examination of a continuous series of editions, such as this, is a necessary task in tracing the evolution of knowledge and understanding the sources of contemporary practice.

The anatomical enlightenment, which Vesalius' books effected, helped to inspire a remarkable development of surgery in the hands of Ambroise Paré and his contemporaries. That generation had learnt its art from the text-book of Giovanni di Vigo, the Pope's consultant, whose *Practica chirurgica* was published in 1514. The book had an unexampled success, probably because it was the only surgery book, as F. H. Garrison pointed out, which discussed the treatment of the two urgent problems of the day —epidemic syphilis and gunshot wounds. It ran through 52 editions

and innumerable translations. In English, four editions of *The Workes in Chirurgerye of John Vigon* were issued during the Tudor reigns, in 1543, 1550, 1571 and 1582. They must have directly influenced the remarkably original group of Elizabethan surgical writers, which included Thomas Gale and William Clowes. English sixteenth-century books are fast disappearing from the market, and the Library has been extremely fortunate in receiving as a most generous gift from Professor John Fulton, M.D., of Yale, a very handsome copy of Vigo's Works " Imprynted by Edwarde Whytchurch," 1550, in a contemporary English binding. The College now possesses each of the four English editions of Vigo's surgery, as well as the Latin text and Italian versions.

Sir Charles Sherrington, O.M., F.R.C.S., whose ninetieth birthday was lately the occasion of affectionate congratulations, has once again made a notable benefaction to the College Library. He has given us several early books, which he used in preparing his picture of the intellectual climate of the sixteenth-century in which Jean Fernel, the father of physiology, flourished. With these is a volume of seventeenth-century documents setting forth the privileges granted to the College of Surgeons of Paris by successive Kings of France, in connexion with their claim for the legal vindication of these rights in 1644. Sir Charles has also given the medal with portraits of Fernel and Paré which he figured as the frontispiece to his book *The Endeavour of Jean Fernel*; the medal was struck after the revolution to record the fusion of medicine and surgery under the Republican health service. Two other medals have lately been acquired, Glasgow University prizes bearing portraits of William and John Hunter. It is sometimes asked why medals should form part of a library's collections. The answer is that traditionally, and with truth, they have been considered, like engravings, to be inscribed documents and so akin to books and manuscripts.

Seventeenth-century anatomy has a strongly physiological bias. Two important accessions to our collection of this period are a copy of Glisson's Anatomy of the Liver (*Anatomia Hepatis*), the Amsterdam edition of 1659, which completes the library's series of this work (1654, 1659, 1665, 1681), and a copy of Descartes' textbook of embryology, *De Formatione Fœtus*, 1672, a work of considerable rarity. Glisson's book is a fundamental contribution to the anatomy and physiology of its subject, and has an outstanding place among the anatomo-physiological works, inspired by Harvey's discovery of the circulation (1628), which, in the course of the century, explored and explained the workings of the body as far as was possible with the technical equipment of the time. Descartes, unlike Glisson, was not a medical man, but in seeking to form a picture of all nature as a base for his philosophy he applied his extraordinarily acute analytical intellect to the functioning of the newest anatomical discoveries, and thus wrote some of the earliest expositions of scientific physiology. Beside these scientific contributions the century produced more popular works of descriptive anatomy, one of
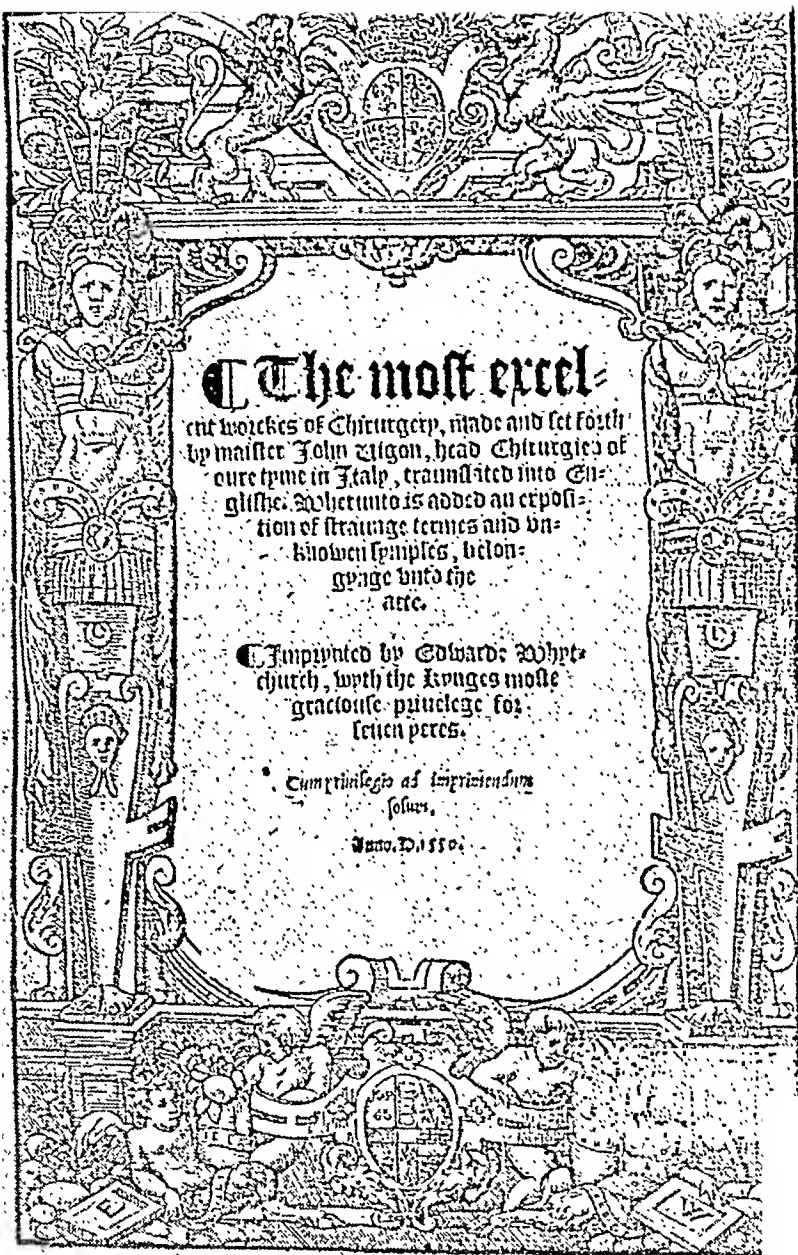
¶ The most excel=
ent worckes of Chirurgery, made and set forth
by maister John Vigon, head Chirurgien of
oure tyme in Italy, tranuslated into En=
glisshe. Wherunto is added an exposi=
tion of strauuge termes and vn=
knowen symples, belon=
gynge vnto the
arte.

¶ Imprynted by Edward: Whyt=
church, wyth the kynges moste
gractouse prtuelege for
seuen yeres.

Cum priuilegio ad imprimendum
solum.

Anno.D.1550.

VIGO, WORCKES OF CHIRURGERY, LONDON 1550
Title page of the copy presented to the College Library by Professor
John F. Fulton, M.D.

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the most remarkable of which was the *Catóptrum Microcosmicum* (" A mirror of the microcosm "), published for Johann Remmelin at Augsburg in 1619. It is an attempt to display on three engravings, by means of superimposed and movable pictures, all the anatomical structures which lie one beneath the other in the human body. Such fragile things as these little hinged pictures necessarily suffer with time, but a copy of Remmelin's first edition has been acquired in very good condition.

The eighteenth-century items are of lesser mark. They include a copy of the privately printed *Lectures* on surgery, 1798, by J. B. de Mainauduc, M.C.S., an Irish pupil of Hunter; Samuel Lee's pamphlet against John Ranby and Caesar Hawkins, serjeant surgeons, concerning his dismissal from the surgeoncy to Chelsea Hospital in 1754, Ranby and Hawkins' defence of their action being already in the Library; and Douglas's Comparative Myology *(Myographiæ Comparatæ Specimen)*, Dublin edition of 1777. An interesting work from the early nineteenth-century is the gift of Mr. W. M. Mollison, C.B.E., F.R.C.S., a beautiful copy of Philibert Roux *Voyage à Londres en* 1814, giving a French surgeon's view of English practice at the end of the Napoleonic war.

Two important collections of more recent date record the work of two very eminent surgeons, Sir James Berry and Sir Arthur Mayo-Robson. Lady Berry has presented Sir James Berry's records of his thyroid and other operations, and the Library has also acquired his collection of monographs and reprints on goitre, giving a survey of the story of surgical intervention on the thyroid from the last quarter of last century till recent times. Miss Violet Mayo-Robson has generously presented a large collection of papers, printed and manuscript, covering the whole professional life of her distinguished father. They comprise a complete series of reprints of his published articles through forty years' active surgical practice, and a very large and interesting correspondence which includes several letters from Lister and many from Moynihan.

DIARY FOR MARCH (15th—31st)

Mon. 15	5.00	Dr. E. M. Darmady—Arris and Gale Lecture—Acute Uræmia ; its Ætiology and Basis for Treatment.*	
Tues. 16	5.00	Mr. A. J. Gardham—Surgery of Malignant Disease of the Pharynx.	
Wed. 17	5.00	Prof. Lambert Rogers—Surgery of the Spinal Cord.	
Thur. 18	5.00	Mr. Rodney Maingot—Surgery of Peptic Ulcer.	
Fri. 19		L.D.S. Examination begins.	
	5.00	Mr. A. Dickson Wright—Varicose Veins and Ulcers.	
Mon. 22	5.00	Sir Reginald Watson-Jones—Fractures of the Spine.	
Tues. 23		Date of Council Election announced.	
	3.45	Mr. R. J. Last—Arnott Demonstration—The Kidneys.*	
	5.00	Mr. M. F. Nicholls—Surgery of the Urethra and Bladder.	
Wed. 24	3.45	Mr. R. J. Last—Arnott Demonstration—The Pectoral Girdle.*	
	5.00	Mr. A. Hedley Whyte—Surgery of the Rectum.	
Thur. 25	3.45	Mr. R. J. Last—Arnott Demonstration—The Knee Joint.*	
	5.00	Mr. T. Twistington Higgins—Urinary Obstruction in Childhood.	
Fri. 26		College closed.	
Mon. 29		College closed.	
Tues. 30	5.00	Mr. R. M. Handfield-Jones—Some less usual examples of Acute Intestinal Obstruction.	
Wed. 31	5.00	Prof. G. Grey Turner—Surgery of the Spleen.	

DIARY FOR APRIL

Thur. 1	5.00	Sir Heneage Ogilvie—Scrotal Swellings.	
Fri. 2	5.00	Mr. R. Scott Mason—Surgery of the Duodenum.	
Mon. 5	3.45	Dr. A. Peacock—The Anatomy of the Nasal Cavity and the Paranasal Sinuses.	
	5.00	Prof. G. Hadfield—Thrombosis.	
	6.15	Dr. Joan Millar—Anæsthesia for Thoracic Surgery.	
Tues. 6		Final Membership Examination begins.	
	3.45	Prof. J. D. Boyd—Development of Pleural and Peritoneal Cavities.	
	5.00	Prof. G. Hadfield—Post-operative Pulmonary Embolism.	
	6.15	Dr. Geoffrey Organe—Endotracheal Anæsthesia and Anæsthesia for Maxillo-Facial Surgery.	
Wed. 7	5.00	Sir Cecil Wakeley—Blood Supply of the Foregut.	
	6.15	Dr. J. K. Hasler—Pre-anæsthetic Treatment.	
	7.00	Monthly Dinner For Fellows, Members and Licentiates.	
Thur. 8	3.45	Prof. G. A. G. Mitchell—The Autonomic Nervous System.	
	6.15	Dr. Herbert Pinkerton—Anæsthesia for Urological Surgery.	
Fri. 9		D. Phys. Med. Examination (Part I) begins.	
	3.45	Prof. F. Goldby—The Trigeminal Ganglion and its Connections.	
	5.00	Prof. R. J. V. Pulvertaft—Epidemic Disease.	
	6.15	Dr. Stanley Rowbotham—Regional Analgesia.	
Mon. 12		Oral Examination in Surgery begins.	
	3.45	Prof. A. B. Appleton—Lung Structure.	
	5.00	Dr. W. Feldberg—	
	6.15	Dr. W. S. McConnell—Anæsthesia for Dental Surgery.	
Tues. 13		Last day for nomination of candidates for the Council.	
	3.45	Prof. W. J. Hamilton—Early History of the Ovum.	
	5.00	Dr. W. Feldberg—	
	6.15	Dr. Francis Evans—Anæsthesia for Rectal Surgery.	
Wed. 14	3.45	Prof. J. Kirk—Kidneys and Ureters.	
	5.00	Prof. D. F. Cappell—The RH Factor—A General Survey	
	6.15	Dr. Howard Bruce Wilson—Anæsthesia for Children.	

*Not part of courses.

Thur. 15	3.45	PROF. H. A. HARRIS—Clinical Anatomy of the Chest.
	5.00	DR. G. POPJAK—Isotopes—Their Biological and Medical Application.
	6.15	DR. JOHN BEARD—Post-anæsthetic Treatment.
Fri. 16		D.M.R.D. and D.M.R.T. Examinations (Part I) and D. Phys. Med. Examination (Part II) begin.
	3.45	PROF. H. A. HARRIS—The Anatomy of Posture.
	5.00	PROF. M. J. STEWART—The Pathology of Peptic Ulceration.
	6.15	DR. WILLIAM W. MUSHIN—Applied Anatomy.
Mon. 19	3.45	PROF. LE GROS CLARK—The Optic Pathways.
	5.00	PROF. D. S. RUSSELL—Intracranial and Intraspinal Tumours.
	6.15	PROF. D. T. HARRIS—Physiology of Respiration.
Tues., 20	3.45	PROF. J. D. BOYD—Derivatives of the Pharyngeal Arches.
	5.00	PROF. D. S. RUSSELL—Intracranial and Intraspinal Tumours.
	6.15	DR. KATHERINE LLOYD-WILLIAMS—Anæsthesia in Obstetrics.
Wed. 21	3.45	DR. G. POPJAK—Isotopes—Their Biological and Medical Application.
	5.00	DR. SCHWEITZER—The Reflex Control of Blood Pressure and Heart Rate.
Thur. 22		L.D.S. Examination (Properties of Dental Materials) begins.
	3.45	PROF. LAMBERT ROGERS—The Cerebral Circulation.
	5.00	DR. SCHWEITZER—Measurements of Cardiac Output and Factors Influencing it.
Fri. 23		D.M.R.D. and D.M.R.T. (Part II) and Primary F.D.S. Examinations begin.
		Voting papers for Council Election issued.
	3.45	DR. F. C. COURTICE—Control of Respiration.
	5.00	PROF. J. H. BIGGART—The Pathology of Head Injuries.
Mon. 26		L.D.S. Examination (Dental Mechanics) begins.
	3.45	DR. F. C. COURTICE—Transport of Oxygen and Carbon Dioxide.
	5.00	PROF. J. H. BIGGART—Some Modern Views on Vascular Disease.
	6.15	SIR GORDON GORDON-TAYLOR—Glands in the Neck.
Tues. 27	3.45	PROF. S. ZUCKERMAN—Male Reproductive Organs.
	5.00	DR. C. R. HARINGTON—Thyroid Physiology.
	6.15	MR. R. MOWLEM—Injuries of the Nose and Sinuses.
Wed. 28	3.45	DR. D. V. DAVIES—Knee Joint.
	5.00	DR. A. C. CROOKE—Adrenal Cortical Hormones.
	6.15	DR. H. A. LUCAS—Nasal Allergy.
Thur. 29	3.15	DR. F. S. GORRILL—The Lymphatic System.
	5.00	DR. M. G. EGGLETON—Modern Methods of Assessing Renal Function.
	6.15	MR. F. W. WATKYN-THOMAS—Tinnitus.
Fri. 30		Final F.D.S. Examination begins.
	3.45	MR. R. J. LAST—Segmentation of Limbs.
	5.00	DR. M. G. EGGLETON—Some Factors affecting Renal Function in Health and Disease.
	6.00	MR. R. G. MACBETH—Osteomyelitis Secondary to Sinusitis.

LAWSON TAIT

by

I. Harvey Flack, M.D.

Assistant Editor, "British Medical Journal"

ARCHIBALD CAMPBELL TAIT, of Dryden, was a writer to the signet who practised in Edinburgh. According to Alban Doran,[1] he was a cousin of the Archbishop of Canterbury of the same name. He married Isabella Stewart Lawson, of Leven, and their second son, Robert Lawson Tait, was born at 45, Frederick Street, Edinburgh, on May 1, 1845. The centenary of his birth was celebrated in Birmingham, his adopted city, just over two years ago.

I have failed to find any official record of Tait's birth. A search of the Register of Births and Baptisms over the period 1843 to 1847 was made for me at the New Register House, Edinburgh, without success. Equally fruitless was a search through the parish records of Edinburgh over the same period which the Scots Ancestry Research Society was good enough to undertake. The explanation for this is not a sinister one. The Registration of Births Act of 1836, which came into operation on July 1, 1837, applied only to England. It was 1855 before the present system of compulsory registration of births came into force in Scotland. A further difficulty arises because in 1843 the Free Church broke away and many people who were involved in this schism would not have their children baptised by the Church of Scotland. Their births were not entered therefore in the only parochial registers which the Registry Office was empowered to take over, those of the Church of Scotland.[2] This accounts for the difficulty of obtaining a statutory record of Tait's birth and it may also help to explain the persistence of the well-known myth of Tait's parentage. Robert Lawson Tait is said to have borne a marked resemblance to Sir James Young Simpson. As Tait's biographer, Stewart McKay,[3] puts it : " the very general opinion at one time was that Tait was the natural son of Simpson." This story grew and spread for several reasons.

In the first place Tait was prone to exaggeration and he often implied[4] that he was much closer to his early teachers, and particularly James Young Simpson, than was probably the case. Secondly, even McKay says that Tait " was not at all displeased with the idea " that he might be Simpson's son. Professor Leonard Gamgee, who has helped me greatly, goes much further and suggests that Tait enjoyed the story and helped to perpetuate it.[5] In these circumstances it was small wonder

that the rumour thrived and was even recalled just before the war by Francis Brett Young,[6] who christened his amusing caricature of a great gynæcologist Simpson-Lyle—a pleasant hyphenation of the rumour and the sugar kings.

The famous rumour is part of the Lawson Tait legend, but it is a rumour and has not a shred of evidence to support it. Such evidence as there is all points the other way, and there is on record a direct denial. This seems to have been overlooked except by W. G. Spencer,[7] who drew attention to an instalment of Mr. T. P. O'Connor's reminiscences.[8] T. P. O'Connor was impressed by Tait's likeness to the statue of Simpson in Princes Street, Edinburgh, and became convinced that the legend was true. He comments : " I need scarcely say that I never dared to put the question to Lawson Tait, though he was a friend of mine, as to whether the legend was well founded or not. He himself, however, volunteered to make that statement to me that the story was not true, and that he came of perfectly respectable, though not distinguished, parents."

Tait's father then was Archibald Campbell Tait, a Guild Brother of Heriot's Hospital. There Tait received his early education and there he won a scholarship which took him to the University of Edinburgh in 1860. He began the Arts course but abandoned it after his first year and never graduated.[9] By 1862 he was watching, if not assisting, James Young Simpson, James Syme, Mathews Duncan and McKenzie Edwards.

In 1866, he qualified as L.R.C.S. and L.R.C.P., Edinburgh, visited Dublin and other schools of medicine, and in 1867 was appointed house surgeon to the Wakefield Hospital.

In 1867, Tait took up his appointment as the only house surgeon at the 12-bed Clayton Hospital, the centre of the Wakefield 'General Dispensary. There were many out-patients to be attended to and a large number of patients were visited in their own homes.[10]

On July 29, 1868, he performed the first of his five ovariotomies as a house surgeon. Tait had seen 30 ovariotomies performed in Edinburgh without a single recovery,[11] and, according to McKay, this being so, it " must have required considerable courage on the part of Tait to tackle his first ovariotomy." This patient, aged 26, died of peritonitis. In May of the same year, 1868, he had opened the abdomen of a woman of 37 only to find a " unilocular ovarian cyst, complicated by general cancer of the peritoneum." This patient survived the operation but lived only for a month.[12] These two cases might have daunted most house surgeons, but Tait went on to record[13] in 1872 what he said himself was " the whole of my experience "—nine cases, including the first fatal one, all the remaining eight being successful.

These figures attracted attention because Tait was unknown and because they were supplied in response to an appeal by the *British Medical Journal* for recent records of cases of ovariotomy. In making this request the *Journal* tabulated the figures available up to that time.[14] Up to 1866, St. Bartholomew's, the Middlesex, King's College, St. George's and

Lawson Tait

I hope mine will go all right.

Yours ever

Lawson Tait

Tait's signature and a reference to the Denholm case.

University College Hospital had had between them 39 cases in which ovariotomy had been performed. There were nine recoveries and 30 deaths. Guy's was more fortunate for there Braxton Hicks had operated on 44 cases with only 21 deaths. Better-still were the results obtained by Mr. Spencer Wells at the Sāmaritan Hospital. Between 1858 and 1867 he had operated on 106 patients with 30 deaths. Then from 1868-71 he had had a further 97 cases with 25 deaths. In other words, Spencer Wells was the outstanding ovariotomist with over 200 cases and a mortality of 27 per cent. Only Keith in Edinburgh had anything like the same experience.

BIRMINGHAM

It was in September, 1870, that Tait acquired the Birmingham practice of Dr. Thomas Partridge. He joined and attended regularly the local medical societies. He was Lecturer on Physiology at the Midland Institute from 1871-79 and his whole-hearted advocacy of the Darwinian theory of evolution attracted attention—and opposition. He wrote regularly for the medical journals and he supplemented his income from practice by writing leading articles for George Dawson. George Dawson[15] was the famous preacher and lecturer. McKay[16] says that George Dawson was " editor of the *Morning Post* " and other authors have copied this curious error.

George Dawson founded and edited, and Lawson Tait wrote leading articles for, the *Birmingham Morning News*, which first appeared in January, 1871, and continued with varying success until January 15, 1876, after which it struggled on as the *Birmingham Morning and Evening News* until May 27 of that year, when it died.[17]

Tait had taken the Edinburgh Fellowship in 1870 and he regarded the practice in Lozells Road as a mere foothold. He soon took rooms in the centre of Birmingham, gave up general practice, and at the age of 26 began practice as a consulting surgeon without holding a hospital appointment. This was early in 1871 and his rooms were in Waterloo Street.

Immediately he began to press for the establishment of a special hospital for the Diseases of Women. In this he was assisted by Arthur Chamberlain[18] and opposed by the *Lancet*.[19]

At length it was agreed that there should be a hospital at No. 8, The Crescent, and that candidates for the office of surgeon must hold the Fellowship of the Royal College of Surgeons of England. Tait had not long been a member of the College but he hastened to London, was admitted a Fellow on June 8, 1871, and returned in triumph to be appointed to the Birmingham and Midland Hospital for Women in July, 1871.

Right from the start Tait showed what the *British Medical Journal* later described as " a want of respect for age and authority remarkable even in Birmingham."[20] He possessed, too, the kind of ruthless courage

which we more often associate with the outstanding surgeons of the pre-anæsthetic era. In my view these are two important keys to his character and to his remarkable success. Once Tait opened an abdomen, if it was humanly possible he went on to complete whatever surgical procedure was necessary. Age and authority might not have sanctioned the procedure. The patient might die and his own reputation might suffer. But, as Alban Doran[21] puts it, " he relied on his good right hand, an excellent principle for any surgeon, provided that, as in the case of Tait, his right hand be really good."

Ovariotomy and Cholecystotomy

Tait began, as in those days all abdominal surgeons had to begin, as an ovariotomist removing large ovarian cysts. Then in February, 1872, he removed an ovary which was the seat of a chronic abscess. Christopher Martin[22] says this operation, the first occasion on which an ovary was deliberately excised for inflammatory disease, took place on February 2. D'Arcy Power gives the same date. Tait[23] himself says he operated on February 11 with " the assistance of Mr. Hallwright and Mr. Bennett May." Later in the same year Hegar and then Battey operated on rather different ovaries for rather different reasons. That Tait's position as the first surgeon deliberately to remove a small diseased ovary is unassailable is shown at length by McKay[24] and concisely by Gamgee, who knew Bennett May well and " never doubted the truth of anything he said." Tait's priority in this connexion was also accepted by Alban Doran.[25]

Tait's next step was to remove both ovaries to " arrest menstrual hæmorrhage due to uterine myoma." On August 1 of the same year, 1872, he cured a case of " perfectly intractable " hæmorrhage by excision of both ovaries.[26] Battey performed the same operation 16 days later for the relief of hystero-epilepsy and in the early days did much more than Tait to publicize this operation.

Tait had already become a figure of some importance in Birmingham and in 1873 he was presented to a larger audience by Sir William Fergusson, who, as President of the British Medical Association, formally awarded Tait the Hastings Gold Medal.[27] His Hastings Prize Essay on " The Pathology and Treatment of Ovarian Disease " appeared in the *British Medical Journal* in seven instalments the following year.[28]

Tait's first abdominal hysterectomy for myoma was performed on January 16, 1874, and reported to the Royal Medical and Chirurgical Society on October 27 of that year. The tumour weighed 11 lb., the patient, aged 34, made a good recovery, and the clamp came away on the eighth day. Spencer Wells at this time had been successful in only three such cases.[29]

Tait at this time was friendly with Spencer Wells. He dedicated his *Diseases of Women* (1877) to him and he was following the Spencer Wells' technique in applying the calliper clamp to the ovarian pedicle.

He had now completed and reported[30] his first 50 ovariotomies with 19 deaths, " operating with the spray and full antiseptic precautions." A different picture was presented by his second group of 50 cases, in which there were three deaths. Tait took the opportunity presented by the completion of his first 100 ovariotomies and read a paper[31] on " The Antiseptic Theory Tested by the Statistics of 100 Cases of Successful Ovariotomy." Tait accepted the germ theory of putrefaction but objected to the results of experiments on dead material being applied to the living patient. The crux of his argument was " that even if bacterium germs lighting on wounds were the cause of much surgical mortality . . . the power of vital resistance by the tissues or the condition of the patient, and the extent and nature of the operation, are of infinitely greater importance as factors in the general result." Meanwhile, as he said, the antiseptic spray was being used as a " royal road to surgical success " by inexperienced and incompetent operators.

It was in 1877 that Tait turned his attention from diseased ovaries to diseased Fallopian tubes. Alban Doran[25] described how " as specimen after specimen arrived at Lincoln's Inn Fields from Birmingham I was astonished to find how salpingitis and allied affections had been overlooked by the pathologist and the surgeon." The pathologists and the surgeons were also astonished—and incredulous.

In February, 1879, Tait made the first abdominal attack on a pelvic abscess. The accepted treatment was by hot douches, rest and conservative measures generally. In a few cases an attempt was made to drain the abscess through a vaginal opening.

Tait's objection to vaginal drainage was that often the abscess could not be reached, or if it could be reached it could not be emptied satisfactorily. He did the simple and obvious thing: opened the abdomen and inserted a glass drainage tube into the abscess. He soon had six consecutive successes to report.[32]

All these patients did well and Tait's conclusion was : " that the opening of such abscesses by abdominal section is neither a difficult nor a dangerous operation : that recovery is made in this way more certain and rapid than in any other : and that in future I shall always advise an exploratory incision where I am satisfied there is an abscess which cannot be reached nor emptied satisfactorily from below."

This was the first occasion on which Tait stressed the " exploratory incision," the indications for which he defined precisely in the 4th edition of his *Diseases of the Ovaries*, the edition he dedicated to Marion Sims. He wrote :

" . . . I venture to lay down a surgical law that in every case of disease in the abdomen or pelvis, in which the health is destroyed or life threatened, and in which the condition is not evidently due to malignant disease, an exploration of the cavity should be made."

Tait's first and successful cholecystotomy was performed on August 23, 1879, and reported[33] to the Royal Medical and Chirurgical Society of

London. This was the first successful cholecystotomy to be performed in this country and the second in the world. A little later, Tait[34] reported on three cases of hepatotomy. Two patients had hydatides of the liver and the third had a large cystic abscess. All three did well.

Ectopic Pregnancy

Tait's next surgical advance is perhaps the one for which he is best known. In his own words[35]:

" In the summer of 1881, I was asked by Mr. Hallwright to see with him in consultation a patient who had arrived by train from London in a condition of serious illness, that illness having been diagnosed by Mr. Hallwright as probably hæmorrhage into the peritoneal cavity from a ruptured tubal pregnancy. . . . This gentleman made the bold suggestion that I should open the abdomen and remove the ruptured tube. The suggestion staggered me, and I am ashamed to have to say I did not receive it favourably. . . . A post-mortem examination revealed the perfect accuracy of the diagnosis. I carefully injected the specimen which was removed, and I found that if I had tied the broad ligament and removed the ruptured tube I should have completely arrested the hæmorrhage, and I now believe that had I done this the patient's life would have been saved."

On January 17, 1883, Dr. Spackman of Wolverhampton had a similar case. The patient was " clearly dying " but Tait " at once advised abdominal section." Describing the case, Tait said : " We got her to bed alive and this is all that can be said." He was bitterly disappointed but he realised the mistake he had made and he learnt from it, which was one of Tait's great virtues.

Less than three months later Dr. Page of Solihull called Tait in to a further case. Tait ligatured the ruptured tube and the broad ligament, and this woman was among the first ever to survive rupture of a tubal pregnancy.

By 1885 Tait was able to analyse his first 1,000 cases of abdominal section.[36] In the course of this report the clamp and Listerism were damned together on the analysis of his 405 ovarian cases. It was a remarkable record and one with three outstanding groups of cases : those concerned with the surgery of the liver and gall-bladder ; those relating to extra-uterine pregnancy ; and those concerned with the surgery of the Fallopian tubes.

The Uterine Appendages

There is no doubt that Tait's success in the surgery of ectopic pregnancy would not have been possible had it not been for his already considerable experience of " the removal of uterine appendages," an experience which in its turn only became possible when he abandoned the clamp.

The last time Tait advocated the use of the clamp was in April, 1879, in a brief " Note on the Principle of Circular Constriction in the Extra-peritoneal Treatment of the Pedicle in Ovariotomy."[37]

The clamp disappeared from his practice so far as the surgery of the ovaries and Fallopian tubes was concerned soon afterwards.

It was at a meeting[38] of the Pathological Society of London, on Tuesday April 5, 1887, with Sir James Paget in the chair, that he showed his specimens of chronic inflammatory disease of the uterine appendages. In his paper[39] describing these specimens Tait explained that in the previous year he had operated on 63 cases of this kind with one death. One specimen had been reported upon by Mr. Bland Sutton, four were in the possession of the gentlemen whose cases they were. He therefore presented 58 specimens from these 63 cases. The mere transport of 58 specimens, many of them from Birmingham to London, must have presented some difficulty, but he knew of the whispers to the effect that Tait's statistics were exaggerated and that no one man could have had so many cases as he claimed. The production of 58 specimens at one meeting was Tait's reply.

Tait's views on removal of the uterine appendages received more than their fair share of publicity and criticism and particularly in 1886 in connexion with the Imlach case. This was the famous case heard at the Liverpool Assizes on August 6 and 7, 1886, before Mr. Justice Cave and a special jury.[40] John Casey and his wife Mary Casey sought to recover damages from Francis Imlach " for performing improperly an operation on the female plaintiff without her consent."

Thomas Frederick Grimsdale, in practice in Liverpool for 39 years and senior physician to the Women's Hospital where the operation had been performed, said that he had had a great many cases of hæmatocœle and hæmatosalpinx and " never had an idea of resorting to the knife." So far as he knew the operation was less successful than the milder treatment by medical attention and rest. It would not have occurred to him to suggest operation in the case of Mrs. Casey.[41]

Later Lawson Tait was called. He said that including abdominal sections, he had operated about 1,600 times. Intraperitoneal hæmatocœle was very distinct from extraperitoneal and was fatal in 96 per cent. of the cases if left long. From Dr. Imlach's description of the case he could say that the operation was a most proper one.

There followed a passage at arms with counsel for the plaintiff which shows well how Tait attracted enemies. Mr. French, Q.C., asked : " Can you explain why Dr. Grimsdale has not had occasion to remove the ovaries ? " There are a dozen possible answers which would have cast no discredit on Dr. Grimsdale, and would not have altered the value of Tait's testimony or the strength of Dr. Imlach's successful defence. Tait chose to reply : " I can point to a large number of cases which have passed through Dr. Grimsdale's hands, which have come to me for

operation, and the explanation is that he has not followed the advances made so as to recognise the value of the operation."

The case had been much discussed before the hearing. Once it was over, however, there were letters and discussions in all the journals on the propriety of what the Americans at least called Tait's operation—removal of the Fallopian tubes and ovaries. The *Lancet* insisted on calling this operation " spaying."[42]

Tait said [43] that he was surprised to find " in the classic columns of the *Lancet* " a continuation of " that extraordinary confusion of terms between ' spaying ' an animal and ' removal of the human uterine appendages '." The two things were done for entirely different reasons and " consequently, to talk of ' spaying women ' is to display great ignorance or to indulge in wilful misrepresentation for the purposes of giving offence." And, for good measure, Tait added : " It will be left to those who cannot recognize the cases when they see them, and who can operate only with a murderous mortality, to talk nonsense about the ' wholesale spaying of women '."

The discussion on " spaying " faded away and is now forgotten but it is worth recalling as a good example of Tait's wrong-headedness. Fundamentally he was right on every point. He was right about the importance and propriety of the operation. He was right in his defence of Imlach. He was right even about the impropriety of using the term " spaying." On all the important points his position was unassailable and he could have warded off any and all attacks quietly and with dignity. Tait was not quiet and had not much use for dignity. In the course of the argument about " spaying " he attacked Grimsdale, Spencer Wells and the *Lancet* with every weapon in his considerable armoury. By so doing he retarded rather than advanced the acceptance of his operation and he gained only so much more unpopularity.

Here again, as on other points, Tait's behaviour was all of a piece. The development of his " removal of the uterine appendages " proceeded so fast because of his indomitable energy, his direct ruthlessness and his pugnacity in the face of opposition. His over-vigorous defence shows all the same qualities expended with great fury on what was essentially trivial criticism that another man might have disregarded. But it is difficult to imagine another man piling up so many cases so quickly and demonstrating thereby the rightness of his solution for this old and neglected problem.

Medical Defence Union

Dr. Imlach was not the first doctor to find himself in legal difficulties and incurring heavy costs. There had been other earlier cases from which the profession learnt, the hard way, that raising funds after the event was not the best way of meeting these difficulties. Some form of union for legal defence was called for and finally the Medical Defence Union was formed. Seeking guidance on the part played by Tait in the formation

of the Medical Defence Union, I approached the present Secretary of the M.D.U., Dr. Robert Forbes. I found him correcting the proofs of a history of the M.D.U. which is due for early publication.[44] He has kindly allowed me to extract what follows on this part of Lawson Tait's activities from his own excellent account.

Soon after its first registration as a company the Medical Defence Union began to set up local branches in Liverpool, Brighton, Manchester, Sheffield and Birmingham. The Birmingham branch was formed on May 6, 1886. Mr. Lawson Tait had been approached by C. F. Rideal, the Secretary, in January, 1886, and invited to join the Union. He did so and he became President. Thanks to Tait's activity confidence in the Union was restored after some preliminary difficulties with the Secretary.

Tait's first conflict with the council of the Medical Defence Union was in 1889. He had received from the Royal College of Surgeons a letter threatening to deprive him of the privileges of Membership and Fellowship, because he had contravened a by-law in signing a document calling a meeting of the Association of Members of the Royal College of Surgeons, of which he was Vice-President. This Association was concerned with the old question of the right of members to participate in the government of the Royal College. Tait wished the Union to support him in a claim for " a declaration of rights, an injunction and damages." At the next council meeting Dr. Robert Saundby protested at the idea of the Union " taking up a faction fight between members and their corporation " and left the room. The other members agreed to support Tait only to the extent of obtaining an interim injunction. At the next annual meeting several members criticised the Council's action and a resolution was passed stipulating that " no part of the funds of the Union should be employed in the political part of the contest between the College and its members."

His final resignation from the Presidency followed a last quarrel about the payment of costs in the libel action which Dr. Denholm brought against Tait in 1892.

Tait and Asepsis

While Tait was presiding over the M.D.U. he was also leading the opposition against antisepsis. F. C. Batchelor[45] said that " he looked upon Mr. Lawson Tait as the great disciple of aseptic surgery." Franklin H. Martin[46] said that Tait " advocated a new theory—' asepticism '." Kellogg[47] describes Tait as "really the father of surgical asepsis." Gamgee[18] says that " Tait was, without doubt, the great inaugurator of aseptic, as opposed to antiseptic, surgery."

I believe that Tait was the first great abdominal surgeon to practise something resembling asepsis even though he did so partly for the wrong reasons. Mathews Duncan who left Edinburgh to take charge of the Gynæcological Department at Bart's only met Tait once or twice but their first meeting was over one of his own cases. After seeing the operation

Dr. Duncan said to Dr. Grigg, who tells the story:[48] "Mr. Tait is a remarkable man. He does his operations in exactly the opposite way to that we are taught, and yet he succeeds in attaining the object we all desire." This was a very shrewd assessment, and exactly the same applies to Tait and antisepsis. Tait did everything in "exactly the opposite way" to Lister and his school and was much more successful than they were in attaining "the objects they all desired."

True asepsis is the logical development of antisepsis. For all of his brief life Tait denied the theory of antisepsis. He agreed that there were germs and he accepted Pasteur's work and Lister's as it applied to dead material in the laboratory. He never believed that it had any bearing on living patients in an operating theatre, and he took a perverse pride in the use of water which was not boiled.[49]

To my mind, therefore, to describe Tait as the "father of surgical asepsis" is illogical. This is borne out by Tait's own 1898 paper [50] on "The Evolution of the Aseptic Method in Surgery." His theme is the same as it always was. "The laboratory fails utterly." " . . . human beings alive differ in their individual results from exactly similar conditions and in ways altogether irreconcilable with the laboratory facts of bacteriology." He followed Syme in his belief in meticulous cleanliness and "at my hands there was no Listerism, no chemical antiseptics, nothing but soap and water." But he kept his hands and his instruments meticulously clean, allowed no talking while he was operating, and allowed no one else to touch the wound. He also preferred the sucker to sponges, which he regarded, correctly, as the "most efficient means of carrying infection."[51] He was in fact practising something like asepsis empirically.

"Asepsis" as Tait and many others construed it at that time meant not "asepsis" as we know it but merely *anti*-antisepsis. Which is probably why Lister himself "looked with disapproval at the change from antisepsis to asepsis."[52]

Fall from Grace

Tait stood for Parliament in 1887 as a Gladstonian Liberal in Unionist Birmingham and was heavily defeated by Mr. Jesse Collings.[53] In the following year he was elected the first Professor of Gynæcology in Queen's College, Birmingham. He had created what was called the "Birmingham School of Gynæcology" and what was originally a derisive title was now "treated with respect."[54] What McKay[55] called "the high-water mark of Tait's career" and what the *British Medical Journal* said was "the zenith of his success"[20] came in 1890. He delivered the Address in Surgery at the annual meeting of the B.M.A. in Birmingham and took a prominent part in the scientific work of the meeting and in the "generous hospitality" of the town. Tait's address[56] was a remarkable one. He was polite, he was logical and he was convincing. He made a plea, which has rarely been bettered, for the training of the surgeon as an artist and as a craftsman.

Then a slip that gave rise to a series of slanders brought this part of Tait's career to an abrupt end. This was the last link in an explosive chain reaction that had been building up over 1892 and the early part of 1893. There was the unhappy Denholm case. Tait's written criticism to a patient's husband of the treatment given her by a colleague caused the first rumblings. There followed the argument about costs with the Medical Defence Union and Tait's resignation of the Presidency. In the background there was his stand on anti-vivisection and every cutting criticism he had made of the men who would not, or could not, see the light as Tait saw it. Then one of his nurses said that Tait was the father of her daughter. Those who knew Tait and admired him believed the nurse, though there is no evidence whatsoever that Tait was ever guilty of anything but this one lapse, and even on that there can be no real evidence.

Tait denied the accusation. He said: "I will spend my last penny in defending myself. Nay, more, my wife is willing to sacrifice her wedding ring to help me in paying the costs." Tait and his wife were a devoted couple, though childless, and they had weathered cheerfully enough the kind of storms that Tait so often aroused. But in Victorian England there was one kind of storm that no professional man, and least of all a gynæcologist, could weather. The accusation was a fact. Whether it was true or false was immaterial. There were scores of people ready and anxious to believe anything of Lawson Tait.

Inevitably Tait's practice fell off markedly and it was no longer true to say of him that "no other man in England controls a larger practice in abdominal surgery."[57] He soon had the additional worry of financial difficulties.

Professor Miles Phillips, to whom I am indebted for a great deal of advice and information, was good enough to pay a special visit to Charles Martin, who is living in retirement in Radnorshire, to discuss some of these points with him. Charles Martin, Christopher's brother, became Tait's private assistant and secretary in 1891 soon after Tait took Christopher Martin into partnership. According to Charles Martin, who stayed there on several occasions, "Buskett Fletchwood," Tait's house at Lyndhurst Road in the New Forest, was perhaps the outstanding example of Tait's remarkable extravagance. It cost about £40,000. Lawson Tait planned to be buried there and got Charles Martin to plant a symbolically prickly shrub (Butchers Broom, *Ruseus aculeatus*) which he desired should be placed over his grave one day. Charles Martin also had to seal scores of bottles of champagne in the wine-cellar. He recalls that Tait was a most generous host, but never in his experience over-drank. Then he suddenly sold everything and retired. He was planning this when Charles Martin left him towards the end of 1893. His financial difficulties were aggravated by the legal action which had been threatened by the nurse and the subsequent falling off in his practice, but were fundamentally due to his own extravagance.

Tait was personally extravagant in his fondness for good food, good wine and good cigars, in his dress, though he dressed for comfort rather than appearance, and in his famous phætons. He entertained lavishly and was never happier than with a house full of guests. He was equally extravagant in his generosity. He often had as many as 20 poor patients in The Crescent when it became his private hospital. This was whenever the Sparkhill Hospital waiting list became too long. He liked the look of Dr. Hallwright's page-boy, so he and Hallwright put the boy into medicine and paid all his fees.[58] All the extravagance had to end abruptly and Tait sold the New Forest house and lived either at " Peterbrook," King's Heath, or at the Yardley Wood bungalow which he kept.

For a time, too, he was not to be seen at the British Gynæcological Society meetings or indeed at any meetings. He even ceased to be a thorn in the flesh of the Royal College of Surgeons. Tait had stood for election to the Council of the College in 1889 and in 1890 when he was narrowly defeated,[59] and again in 1891 when he polled more votes than in the previous two elections.[60] He was supported by the Association of Fellows and campaigned vigorously for representation of members on the Council and for a larger representation of provincial Fellows. The Council was, he said, " too metropolitan " in its constitution. For a while he even stopped writing to the medical journals, though not for long.

Much of the fire had gone out of Tait, but the extraordinary thing is that he did come back. A lesser man might have bowed his head and taken to his books or his garden. Tait reappeared in all the medical societies. There was a change in him, however—a change which I think may have marked the beginning of his illness.

It was on the subject of hysterectomy in 1892 that Lawson Tait showed the first evidence of the kind of conservatism against which he had been battling himself since he left Wakefield. The voice is the voice of Lawson Tait but the sentiments are precisely those which most exasperated him as a young man.

It was at a meeting of the British Gynæcological Society on Thursday February 11, 1892. Dr. Heywood Smith[61] read a paper on subperitoneal hysterectomy. He described three cases in which he had closed the peritoneum over the stump of the cervix, a procedure which he thought would prove " more scientific than the abdominal fixation of the stump." Mr. Lawson Tait[62] did not agree. " . . . it was amusing—positively amusing—to hear men who had had only four or five cases talking about the ease with which this or that could be done . . . with an experience of many hundred cases he solemnly asserted that in the absence of some great discovery—of a kind not as yet even hinted at—the intraperitoneal method was impossible for general adoption. . . . Whether therefore the clamp method was crude or not it was at present the only method susceptible of general application . . . short of some altogether fresh light

upon the subject he intended in future to adhere to the extraperitoneal method."

Contrast this with the Lawson Tait who six years before had criticised Granville Bantock. Bantock[63] had been discussing uterine fibroids and their removal. He said : " that in every case in which he had trusted to the ligature a fatal result had ensued, and he would not resort to it again ; while in similar cases treated by the *serre nœud* he had not had a moment's anxiety."

Bantock was reflecting the majority view at that time. Ligatures were not trusted and the usual practice was to apply whichever clamp the operator favoured, and to leave the firmly clamped stump outside the abdominal wound. The newly elected President of the British Gynæcological Society, Mr. Lawson Tait,[64] admitted that the question was a difficult one, but he did not think that Dr. Bantock was right when he said that he would not employ the ligature so long as he had the clamp— " at any rate, all that he (the President) could say was that the intraperitoneal method of dealing with the pedicle would have to be employed if we are ever to get results as good as those in ovariotomy." He thought that " by patient perseverance " similar satisfactory results would also be achieved in hysterectomy, " but he was quite clear it would not be by the use of the clamp."

In both cases this was Lawson Tait speaking. The man whose own success rested in great part on the abandonment of the extraperitoneal clamp method in ovariotomy and the substitution of the intraperitoneal method. Is it too fanciful to suggest that by 1892 either the disease which ended his life had already begun to affect his outlook and to alter his previous direct and clear-headed approach to any problem of surgical technique, or perhaps that financial, legal and other worries were already beginning to distract his attention ?

Tait's Illness

It seems generally agreed that Tait was suffering from nephritis by May, 1893. It may be significant that he made his will on April 6, 1893, leaving everything to his wife. A period sidelight on this document, which could not well be shorter or simpler, is that one of the witnesses is Amy Rankilor who describes herself as a " Typewriter." If April or May, 1893, is accepted as the time when Tait realized his condition and had perforce to restrict his activities, then I think it not unreasonable to suggest that his Spencer Wellsian views in February, 1892, on Heywood Smith's subperitoneal hysterectomy were not those of a man in Tait's normal robust mental and physical health.

By 1895 however, he was doing everything that he had done before. He was addressing medical societies and writing letters and papers. There was a letter[65] on " A Rare Condition of the Omentum." Another characteristic one was on " the exceeding rancour with which Mr. John Frederick Bullar persecutes his next-door neighbour at Southampton."[66]

There was a paper defending his " Stafford Knot."[67] He was in demand again as an operator. He went over to the South of France to operate on Professor Pierre Budin, who was suffering from some obscure hepatic disorder.[25]

He still read papers before the Birmingham and Midland Counties Branch of the British Medical Association and patients still came to him from as far away as Barbados[68] to have their renal calculi removed. He wrote his paper[69] on the method of lumbar colotomy McKenzie Edwards had taught him, and he described a successful application of what the Americans called the Tait-Porro operation for unavoidable hæmorrhage at term.[70] Christopher Martin[53] describes this as his " last important article."

This last paper in the *Lancet*, " The Treatment of Unavoidable Hæmorrhage by removal of the Uterus," was short and to the point. The operative details had been described by Tait[71] a long time before. His rebuttal in advance of the " argument of the brothel-keeper " which the " mutilationists " would be sure to put forward is familiar and follows the lines of the " spaying " controversy. What was new was the suggestion that hysterectomy should be considered in cases of placents prævia.

Tait's last letter to the *British Medical Journal* was a characteristically wrathful outburst[72] against Dr. Robert Saundby, then Chairman of the Council of the B.M.A., written on June 1, 1899.

Two days later, on June 3, Tait replied for the visitors at a luncheon after the opening of a new railway station at Droitwich. He said :[73] "I came to Droitwich last night ; I was quite well ; I had a bath and to-day I am better." He was taken ill soon afterwards[74] and his death certificate states specifically, " Nephritis. Uræmia—10 days." Here, as at every stage of Tait's life, there is a story. Tait was usually attended by Dr. Woodhouse of Llandudno. He was away and a young locum, Dr. H. F. B. Williams, was in charge. He was worried about his responsibility and asked Tait if he would like another opinion. Tait said " No" and looked at the cigar he was smoking: " I am smoking a very good Laranaga—and the last I shall ever smoke."[75] He died that afternoon, June 13. His body was cremated on June 16 at Anfield[76] and the ashes were afterwards placed in an urn in Gogarth's Cave, an ancient burial place in the grounds of his Welsh home.

Conclusion

My interest in Lawson Tait was first aroused ten years ago and I wrote then : " In England, Robert Lawson Tait did more than anyone else to make gynæcology a specialty."[77] I still think that is a true assessment, but trying to encompass Lawson Tait in a few words is difficult. The difficulties are not lessened by the extraordinary differences in the literature about Tait.

Guthrie,[78] admittedly more concerned with medicine than surgery, dismisses Lawson Tait in a few lines as " a pupil of Simpson in Edinburgh who built up a large practice in Birmingham." Cuthbert Dukes[79] contrived to write his life of Lord Lister without mentioning Tait. This is at least more polite than Jameson[80] who says that Lawson Tait's success " as judged by *his* statistics, was marvellous," and goes on, " Tait was domineering, frequently uncouth and coarse, and had a highly exalted opinion of Lawson Tait." Jameson also mentions the notes about Lawson Tait in Franklin Martin's extremely naïve and highly inaccurate autobiography, and refers to Tait's " one-sided controversy with Lister."

That is one side of the medal—either no mention, brief dismissal or critical mention. The other side, by contrast, is quite startling. Stewart McKay[81] dedicated his book on " Ancient Gynæcology " to Lawson Tait. In his preface he wrote : " Mr. Lawson Tait, my teacher and friend . . . added to the advancement of operative gynæcology more than anyone else who has preceded him ? This man was a genius. . . . He had a brain that was omniscient. . . ." The same note runs right through his biography of Lawson Tait and prompts his quotation of William J. Mayo's reference to Tait as " the father of abdominal surgery " ; and Joseph Price's remark : " In pelvic surgery Tait stood first and taught us the best we know. He has had no very close second. . . ."

So in considering Tait we are faced with one group of surgeons and gynæcologists who thought he was a genius and could do no wrong. In opposition are the detractors, who grudgingly admit only his technical skill and paint a thick black picture of his quarrels, his wrong-headedness and his private life. It is temptingly easy to assume that a real assessment of Tait lies midway between these two extremes. The too facile assumption is that both sides exaggerated. His followers exaggerated his qualities and his success, and his opponents exaggerated his defects.

In an attempt to resolve this dilemma I tried to read everything Tait had ever written and most of the things that were written at him or about him. At first I seemed to be collecting contradictions. Tait was mean about notepaper and absurdly generous about far more important things. He would charge a private patient an outrageous fee and spend it all on Japanese curios. He would fill his private hospital with patients who had not a guinea between them and then force into bankruptcy the first secretary of the Medical Defence Union for a debt that he could have paid himself without even noticing it. He was a sound antiquarian and in favour of universal free education. He simplified surgical technique and complicated for years the Listerian issue. His operative skill was remarkable and his arithmetic was poor. He perfected a method of repairing the torn perineum and would never repair a torn cervix, probably because he disliked Emmett. He loved a losing cause and hated the counsels of age and experience. In many ways a complete authoritarian he never heeded the accepted authorities. He was kindly and generous and a most bitter enemy. He was brilliant in debate and more likely to

be successful when he was wrong than when he was right. He could be completely charming or unbelievably rude. An Irish nurse who was as rude to him as he had been to her delighted him. A telephone that annoyed him was torn from the wall and jumped on. He would write a long letter about daffodils[82] in the morning and a bitter tirade about " obstetric physicians " in the afternoon.

He was surgically brilliant and bacteriologically impossible. He admired Semmelweiss and his " I attribute my favourable results simply to the fact that I operate with clean hands."[83] And he had no more faith in the theory underlying the phrase·" clean hands " as Semmelweiss used it than he had in the surgical application of the work of Pasteur and Lister.

These are not contradictions that cancel each other out. They are all part and parcel of one remarkable man. The true picture of Lawson Tait does not lie somewhere between the portrait of a blackguard painted by his enemies and the pastel of a surgical saint depicted by his disciples. Lawson Tait was a completely ambivalent character and many of the qualities that made him an outstanding surgeon served also to make him on occasion an impossible individual. Much of his surgical success was due to his courage and his ruthlessness. It was generally felt that women with ruptured ectopic pregnancies should be allowed to bleed to death in comfort. Lawson Tait operated ruthlessly and courageously and was in no way dismayed by the death of his first case.

He did the simple and obvious things superbly well, using a minimum of instruments and an absurdly small incision. He made pelvic surgery a separate discipline and he demonstrated clearly that the liver and gallbladder were as surgically approachable as the ovary and the uterus. He did much more than any other single individual to create a gynæcological specialty and to blaze the trail for the great generation of abdominal surgeons that followed.

He did all this in 20 crowded years between 1870 and 1890. And over the same period he quarrelled with so many people that his quarrels are remembered and the great contribution he made to the advancement of pelvic and abdominal surgery is too often forgotten.

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PROGRESS IN THE SURGICAL TREATMENT OF CARCINOMA OF THE ŒSOPHAGUS AND UPPER STOMACH

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I WOULD HAZARD the guess that it is the secret desire of most American surgeons to be invited to lecture before this great College, respected and admired the world over for its great tradition and high position in the surgical world. I am not unmindful of the honour you have accorded me. I feel as though I were bringing coals to Newcastle in discussing the subject of cancer of the œsophagus with you, because British surgery has been foremost in the development of this difficult field. Many of your leading surgeons have made notable contributions to the subject and I would like to pay tribute to Grey Turner, Ogilvie, Abel, O'Shaughnessy, Tanner, Lewis, Raven, Thompson Taylor and others.

One can liken the growth of œsophageal surgery to the growth of a child. The period of gestation of this infant occurred in the latter part of the nineteenth century and the early years of this century. The subject captured the imagination of such men as Billroth, Biondi, Czerny, Kümmel, Sauerbruch, Voelcker, Gosset and others and isolated attempts were made to extirpate mainly cancers of the upper stomach without much success. The child was born with a lusty cry in 1913 when Franz Torek of New York for the first time successfully resected a cancer of the middle of the thoracic œsophagus. Then followed a rather marasmic infancy of this child with infrequent attempts, made mainly by Grey Turner, Lilienthal, Eggers and a few others, to solve the difficult problem. The years 1936 and 1937 mark a turning point in the growth of this branch of surgery because then, for the first time, three consecutive successful resections were reported and Phemister carried out the first trans-thoracic œsophago-gastrostomy. Since then this child has grown into a robust adolescence and gives every evidence of rapidly increasing development.

In this paper I would like to indicate in a general way observations based on an operative experience with approximately 225 cases, emphasizing particularly the progressive steps that have been taken in the development of this branch of surgery and the changes that have occurred in the preoperative, operative and postoperative management. Progress in any field can take place only by a process of trial and error

and this is particularly applicable to the subject under discussion. It must be emphasized that the great progress in œsophageal surgery during the past 10 years can in large measure be attributed to the development of the finer technical details of gastro-intestinal surgery during the past 25 years, the understanding of the altered physiological relationships attending open thoracic procedures, the rapid studies in the field of anæsthesiology and the recent discovery of the new antibiotics.

It has been repeatedly demonstrated that early diagnosis constitutes one of the most important phases of the therapy of this disease. Early ulceration of the œsophageal wall or minimal diminution in the calibre of the lumen will produce symptoms more quickly than will similar aberrations in any other part of the gastro-intestinal tract. It therefore becomes important to view with concern any sudden change in the function of deglutition and to adopt measures to establish a positive diagnosis. It is unnecessary to stress the importance of a thorough radiographic examination of the œsophagus, cardia and the so-called silent area of the stomach along the beginning of the greater curvature. In my opinion, every patient with radiographic evidence suspicious of carcinoma should be œsophagoscoped. It is sometimes extremely difficult to differentiate a benign from a malignant lesion on the radiographic evidence alone. I have seen smooth conical obstructions at or near the cardia which indicated probable benign cardiospasm. Yet the biopsy specimen obtained at œsophagoscopy disclosed the presence of a neoplasm. On the other hand, it is not uncommon to encounter instances where the X-ray evidence suggests strongly a neoplasm, and yet, the œsophagoscopic examination discloses a benign stricture, a peptic œsophagitis, or a chronic inflammatory reaction as seen in syphilis. These are some of the important reasons for routine œsophagoscopy and biopsy in establishing a diagnosis of carcinoma.

The pathologist's report of squamous cell cancer from the biopsy specimen indicates that the tumour has originated in œsophageal mucosa. When the finding is one of adenocarcinoma, the indication is clear that the tumour has had its origin at the cardia or upper stomach. We have encountered many examples of extensive upward growth along the œsophageal wall of tumours arising at the cardia. This extension may be entirely submucosal and may not be evident to the œsophagoscopist. Growth of squamous cell carcinomas in the opposite direction apparently does not occur. Such tumours will grow as far as the œsophago-gastric junction and will not proceed beyond this point. This has been a curious pathological differentiation between these two types of cancers.

Increasing operative experience during the past 10 years in the treatment of this disease has disclosed certain pathological information concerning lymph node spread which was not known heretofore. Until recent years, all our knowledge concerning the pathology of this disease was based on autopsy material and represented the terminal stages. It is now known that some of the tumours of the thoracic œsophagus may grow in a peripheral

direction and quickly become inoperable by reason of fixation to the aorta, hilus of the lung, left main bronchus or vertebral column. On the basis of this operative experience in the various stages of the disease, it has also become clear that spread to the lymph nodes may be not only to the immediate vicinity of the tumour but also to nodes far removed from the growth. That is to say, tumours of the middle third of the œsophagus may spread to the paracardial or peripancreatic nodes below the diaphragm as well as to the regional nodes in the mediastinum or hilus of the lung or proximally to the lower cervical region (node of Virchow). Adenocarcinoma arising at the cardia or upper stomach may show no extension below the diaphragm, yet exploration may disclose extensive dissemination to the structure in the chest, viz., mediastinal nodes, pleura, lungs or pericardium. Squamous cell tumours rarely, if ever, metastasize to the liver. However, hepatic involvement via the portal system is frequently seen with adenocarcinoma of the cardia.

Increasing experience with the surgical treatment of this disease has demonstrated very clearly that it is desirable to consider it as a group co-operative problem to include the combined efforts of roentgenologist, œsophagoscopist, internist, anæsthetist, surgeon, operating-room staff and nurses for postoperative care. This experience has also emphasized the value of careful preoperative preparation, frequently prolonged for two or three weeks. A study of the postoperative complications and causes of mortality has shown the great preponderance of cardio-vascular accidents over all other complicating factors. Postoperative cardiac difficulties or cerebral accidents are in the main in the imponderable group and cannot usually be predicted preoperatively or even guarded against. However, the internist becomes an important member of the team in his appraisal of the cardiovascular capacity of the patient to withstand an extensive operative procedure. The incidence of postoperative pulmonary complications has been materially decreased since we began to utilize preoperative nebulization of penicillin of the bronchial tree. This has been a real advance in the preoperative preparation. The low incidence of chest and wound sepsis in our series is indicative of the great importance that we attach to scrupulous wound protection, non-traumatic operative technique, thorough hemostasis and meticulous suture anastomosis. This extra effort at the operating table will pay dividends in the form of a low wound and chest morbidity.

It seems superfluous to stress the importance of meeting the patient's protein, carbohydrate, fluid, electrolyte and vitamin requirements during the period of preoperative preparation. Deficiencies, as indicated by blood studies and clinical appraisal, are made up by intravenous injections of proteins, plasma, whole blood transfusions, parenteral vitamins, etc. Checking of the patient's weight by daily measurements aids the surgeon materially in determining the degree of nutritional improvement. Repetition of the original blood studies in the latter part of the preoperative period is also desirable for comparative purposes.

The progressive improvement in the operative management of cancer of the œsophagus and cardia may perhaps best be emphasized in the form of a step-by-step tabulation.

1. There seems to be fairly general agreement now that the question of anæsthesia is of paramount importance. I am convinced that, unless the anæsthetist is thoroughly competent, the surgeon should not undertake this operation. In talking to many surgeons who have attempted œsophageal resections in appreciable numbers, I have been impressed with the fact that many of them have been severely handicapped by serious anæsthesia problems. This has been particularly noticeable with the foreign surgeons, who are now visiting America in increasing numbers. The anæsthesia of choice to-day, on the basis of an extensive experience with various modalities, is intratracheal gas-oxygen-ether.

2. For cancers of the middle third of the œsophagus it is no longer necessary to utilize the Torek operation. It is now possible to restore normal gastro-intestinal continuity by the operation of supra-aortic œsophago-gastrostomy, originally described by me in January of 1944. By an accurate ligation of the blood supply of the stomach, i.e., the vasa brevia, the left gastro-epiploic and left gastric artery and leaving atttached to the stomach the gastro-epiploic arch, one can mobilize the organ to such an extent that it can be brought to the apex of the chest without jeopardizing its vitality and anastomosed to the stump of the œsophagus.

3. When an operable cancer is found in the region of the superior mediastinum, i.e., the region from the upper surface of the aortic arch to the apex of the chest, it is possible to bring the carefully mobilized stomach to the apex of the chest, pull out its upper end into the neck and anastomose it to the œsophageal stump in the cervical region. Recently I was able to demonstrate that this is entirely feasible and that the blood supply of the stomach remains intact. The utilization of the stomach in effecting these high anastomoses has made unnecessary the various complicated plastic procedures which employed tubes of stomach wall and loops of jejunum in an attempt to restore œsophago-gastric continuity. The operation of supra-aortic œsophago-gastrostomy is a recent development in œsophageal surgery.

4. Since Phemister's first successful œsophago-gastric anastomosis in 1937 for cancer of the distal part of the œsophagus, most surgeons have adopted this procedure as a routine measure for tumours of the lower œsophagus and upper stomach. Until recently, this was accomplished by a transthoracic transdiaphragmatic route. Frequently, after the patient had been subjected to a formidable transthoracic exploration an inoperable tumour was disclosed by reason of extensive metastases below the diaphragm. It is for this reason that I suggested some years ago the great desirability of demonstrating a resectable tumour by the simpler expedient of an abdominal exploration alone. If the growth was found operable, the abdominal wound was closed and a transthoracic resection was then done. A recent development of this thought has been the

perfection of a combined abdomino-thoracic incision with simultaneous exposure of both the upper abdomen and the left thoracic cavity. This incision has simplified in no small measure the whole problem of the surgical treatment of cancer of the lower œsophagus and upper stomach. Because the incision is a large one, the approach is more direct and all operative manœuvres can be carried out under direct vision with minimal trauma. This has been clearly discernable in the much smoother post-operative course, the lower mortality and decreased incidence of post-operative complications. The combined abdomino-thoracic approach has a wide field of applicability and should be the exposure of choice for total gastrectomy.

5. Increasing experience during the past five years has effected some changes in the technical details of these operations. The important ones may be mentioned briefly :

a. The left leaf of the diaphragm should be put at rest by pinching the phrenic nerve above the diaphragm.

b. It is not necessary to apply clamps to either the œsophagus or stomach in order to minimize contamination. The œsophagus may be kept empty by an indwelling Levin tube during the operation. The stomach can be emptied by suction.

c. In the performance of the anastomosis, it has been clearly demonstrated that interrupted silk sutures should be used if one is to avoid a stricture. I am inclined to agree with Sweet that the excision of a button of gastric wall the approximate size of the œsophageal lumen also aids in the prevention of stricture.

d. Slight telescoping of the suture line by drawing the stomach over it and anchoring of the stomach to both edges of the mediastinal pleura will prevent drag on the suture line.

e. It is important to anchor the diaphragm around the transplanted stomach in such a way as to prevent herniation of abdominal contents into the chest.

f. We have never found it necessary to use an indwelling Levin tube during the post-operative period. In fact, there may be some danger from pressure necrosis in the suture line.

g. There seems to be general agreement that under-water drainage of the chest for at least a few days postoperatively is desirable.

h. Before closure of the operative wound, 50,000 to 100,000 units of penicillin should be injected into the pleural and abdominal cavities. It probably has some local beneficial effect.

The postoperative care of these patients is concerned mainly with the early detection of chest complications and the immediate application of the necessary therapy. Collections of trapped air should be aspirated as quickly as possible. Serious respiratory and cardiac difficulties may ensue if this is not done. Oxygen therapy should be utilized for the first day or two. Swallowing is interdicted until the fourth day when sips of water are permitted. The fluid intake is increased rapidly thereafter.

Soft food is usually given on the seventh or eighth day. In the last two years we have given penicillin parenterally during the early postoperative period. The latter has probably been a large factor in the reduction of pulmonary complications.

The problem of palliative surgery in cancer of the œsophagus and upper stomach is one open to considerable discussion. The question resolves itself into whether or not a surgeon is justified in subjecting a patient to an extensive resection in the presence of non-resectable local and distant metastases which would preclude any possibility of a cure. Some surgeons feel that palliative surgery of this sort is justifiable solely for the purpose of restoring the act of swallowing if only for a few months. The majority of surgeons, I believe, feel that the risk is too great and that the results do not justify the effort. I bring up this much debated question because it is important for surgeons, when they report a series of cases, to indicate most clearly which resections are palliative and which are otherwise. If all surgeons adopted a similar plan of reporting their operations for cancer, a great mass of valuable material could be collected and a clearer picture could be obtained of the value of surgical therapy as opposed to other methods.

Progress in this field of surgery has been so rapid in the past 10 years that it can be safely predicted that the next decade holds great promise of a steadily decreasing operative mortality and a rapidly increasing number of long-term survivors.

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TOOTH FORM AND FUNCTION

Lecture delivered at The Royal College of Surgeons of England

on

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by.

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TEETH ARE SPECIAL modifications of the epidermis at the entrance to the alimentary canal developed for the dual purpose of seizing food and preparing it for ingestion. Invertebrates, subsisting for the most part as they do on a diet which is either fluid, soft or finely particulate, have no need for teeth and it was only with the evolution of vertebrate animals feeding on solids that true teeth appeared. Like skin itself and other structures derived from it such as horns and teeth they consist typically of a superficial covering derived from ectoderm (the enamel) and a basic structure of mesodermal origin (the dentine &c.). Like skin too they are semi-deciduous and undergo periodical replacement, in lower vertebrates regularly, in higher vertebrates periodically. As evolution proceeded and the life of the individual teeth became more prolonged they tended to become more specialised in shape and structure to serve functional needs and at the same time to be attached to the jaws by a mechanism more complex than the simple embedding of an expanded base in the subcutaneous connective tissue which serves to secure the teeth in selachian fish.

In these primitive vertebrates the homology of the teeth with the horny scales of the skin is obvious, but even here there is a clear distinction in form between predatory sharks, whose sharp edged teeth have as their main function the seizing of their prey, and skates and rays whose blunt or flattened teeth, closely set in a semi-cylinder, serve to crush and prepare for ingestion the immobile substances on which they feed : in both the life of the individual tooth is to be measured by months. When true bony fish came to be evolved their variety of feeding habits was accompanied by greater variation in the attachment and structure of the teeth, but these remained for the most part of simple conical form (homodont) with a brief life and frequent replacement (polyphyodont). In these fish teeth are not confined to the jaw margins but often appear on the palate and the gill-arches where they serve the function of food ingestion by pushing the captured prey down the gullet with rhythmic movements of the gills. A contrast in function and form is shewn by the pike and the wolf-fish (Fig. 1). The teeth of the pike are numerous and sharp, with those on the jaw margins and gill-arches ankylosed, those

on the palate so hinged that the very movements of the captured prey tend to turn it towards the axis of the gullet down which it then wriggles its own way, assisted by the teeth on the gill-arches. The wolf-fish feeds on molluscs and bivalves which are plucked from their attachment to the rocks by the curved front teeth and then crushed in preparation for swallowing by the blunter back ones.

In most reptiles the teeth are replaced at frequent intervals and the form remains a simple one. The poison fang of the viper was evolved from the simple grooved cone of the water-snake's fang to a closed canal which injects the poisonous parotid secretion like the hypodermic needle whose design it is said to have suggested : this is perhaps the most perfect correlation of form and function in the whole history of tooth design. Equally interesting is the appearance in the extinct order of theriodont reptiles of that differentiation between the form of the front teeth for seizing food, and the back teeth with their function of preparing it for ingestion (heterodont dentition) which we have seen foreshadowed in the wolf-fish and which was to be the main characteristic of the mammalian dentition. In these theriodont reptiles there is also evidence that tooth generations had been reduced to two as in the mammalia.

Early mammals had less specialised dentitions than we see to-day. Far more of them achieved the standard formula of 44 teeth, Incisors $\frac{3}{3}$ Canines $\frac{1}{1}$ Premolars $\frac{4}{4}$ Molars $\frac{3}{3}$ (now possessed by only five species including the pig, the horse and the mole) ; these are conveniently divided for description into " front teeth " and " check teeth " and their arrangement suggests an omnivorous diet. In fact the arrangement of the teeth as a single row with the front teeth sharp for seizing food and the blunter back ones a continuous surface suitable for crushing it in preparation for swallowing—this is the basic design of the mammalian dentition from which more specialised forms have evolved. In animals, however, that remained omnivorous like most marsupials, primates and insectivora no great departures from this design have taken place and man's dentition, like his anatomy generally, remains primitive. It is significant in this connection that amongst existing orders of mammals only marsupials, primates and insectivora are as old as early eocene time (Fig. 2). From the primitive pattern of early mammals dental development has been conditioned mainly by diétetic specialisation, though sexual selection and what may be termed evolutionary momentum have played their parts, and we may now examine different diets and their influence on tooth form.

An insectivorous diet if pure may lead to the evolutionary loss of all teeth as in the ant-eaters. Most animals in the order of insectivora are, however, partly omnivorous and display the dentitions typical of primitive mammals save that the cusps of both front and cheek teeth are more sharply pointed. Animals such as the pig family are also omnivorous and have teeth of the primitive pattern except that their canine tusks originally evolved perhaps for grubbing food from the ground have taken

Upper Teeth of Pike   Lower Teeth of Pike   Upper and Lower Teeth of Wolf-Fish
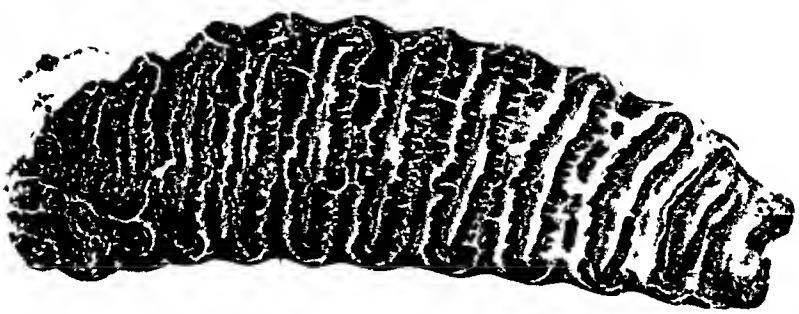
Fig. 1.   Teeth of Fishes.

Phalanger   Tarsius (Order Primates)   Mole
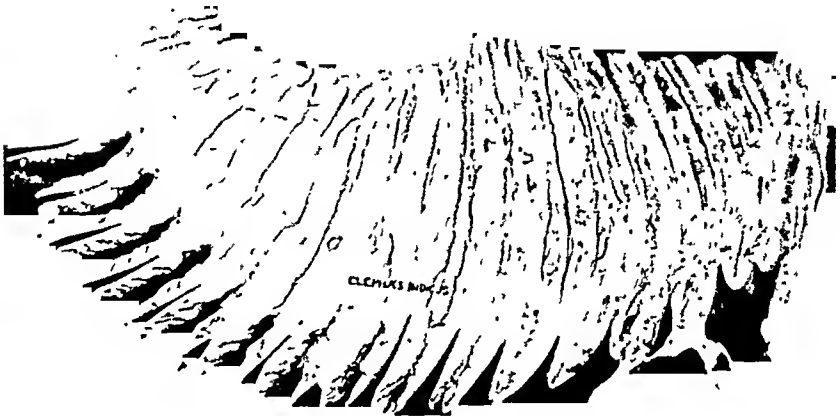(Order Marsupalia)   (Order Insectivora)

Fig. 2.   Upper Teeth of Omnivorous Mammals.

Occlusal Surface, Molar of Indian Elephant

Vertical Section, Molar of Indian Elephant

Skull of Rabbit with Outer Alvelous removed
in the Mandible

Fig. 3. Teeth of Herbivorous Mammals.

Cat   Dog   Badger

Fig. 4.   Upper Teeth of Carnivora.
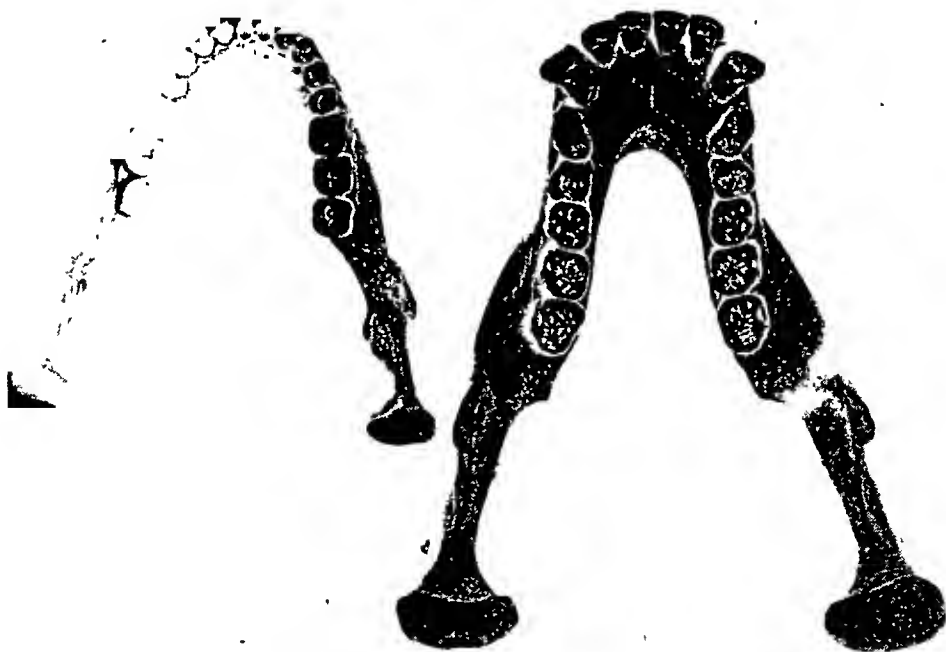
Fig. 5.   Mandibles of Man and Orang Utan.

Otter

Sea-Otter

Fur Seal

Seal

Dolphin
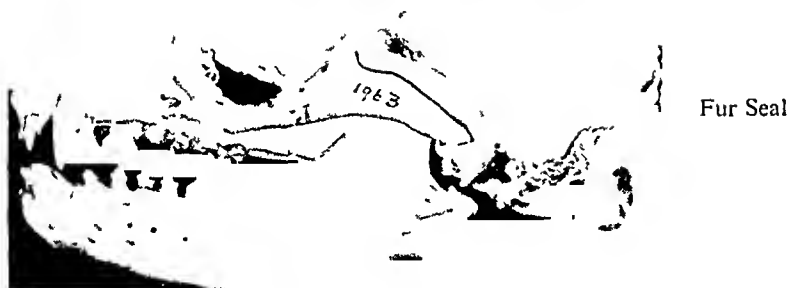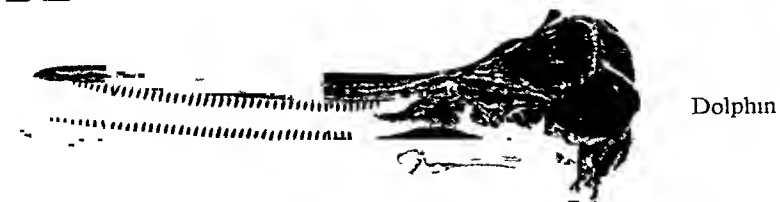
Fig 6   Teeth of Piscivorous Mammals.

on a secondary character as sexual weapons and in the male are of persistent growth and keep perpetually sharp. A true herbivorous diet requires more mastication than any other to break up the cellulose capsules before digestion can proceed and entails maximum wear of the cheek teeth. On the other hand, such food is easy to seize and herbivorous animals therefore tend to a reduction in the number and size of the front teeth and a development of the cheek teeth. The former trait is illustrated by the edentulous premaxillae of pecora (true ruminants), and the problem of masticatory wear is solved along several different lines. Primitive cheek teeth like human molars had shallow crowns and long roots : such teeth lose first their cusps and then their crowns if subjected to coarse granular diet as we may see in primitive races of mankind. So herbivorous ruminants developed cheek teeth with deep crowns and roots relatively short (hypsodont teeth) which allowed plenty of room for wear. Another development—which we can observe throughout its course in species with a well documented fossil ancestry like the horse and the elephant—modifies the structure of the tooth. The tooth crowns develop high ridges clothed with thick enamel which is also folded deeply into the crown substance. Finally coronal cementum fills up the spaces between the ridges so that however much the crown becomes worn its surface is always rough enough to be a functionally efficient mill exposing three calcified tissues, enamel, dentine, and cementum, each with a different rate of wear. The horse is a good example but perhaps the best is the large compound molar of the elephant. This vast mammal has only a single pair of opposing teeth on each side ; these are periodically replaced but the same tooth can render efficient service for more than sixty years. Herbivorous rodents like the hare and the beaver have molars of persistent growth, the most satisfactory of all solutions of this problem except the perennial replacement of teeth enjoyed by fish and reptiles, a device-discarded by mammals (Fig. 3).

A carnivorous diet demands a dentition specialised in precisely the opposite direction to that just described, for flesh requires no mastication other than its division into sections small enough to be swallowed and the molars of the cat are reduced to a single pair on each side, of which the upper is rudimentary while the lower is the large carnassial blade which, opposing the similar blade of the last upper premolar slices flesh into pieces of suitable size. The front teeth, on the contrary, are highly developed for seizing prey, and in particular the large canines, equally developed in both sexes, are characteristic of carnivora. Only a minority in this order, however, are pure flesh-eaters : some like the dogs while subsisting mainly on flesh will tackle bones and have more molar development than the cats, while others like the badger and most bears are omnivorous with dentitions approaching the undifferentiated omnivorous pattern. Animals in other orders which adopt carnivorous habits tend to develop the characteristic dentition and the Thylacine or Tasmanian

wolf is such a one, a marsupial whose skull and teeth could well pass on casual inspection for those of a dog (Fig. 4).

Mammals were evolved for life on dry land; but many have reverted to the water and a piscivorous diet, and they provide a fascinating picture of evolutionary change. The most suitable dentition for such a diet is that of a predatory fish, an undifferentiated row of simple sharp conical teeth and all stages in the journey back to this, the dental base line of the vertebrates are illustrated: those mammals whose ancestors began the reversion earliest in time approach it the most closely. The otter equally at home on land and water has the teeth of a typical carnivore of the bear group. His cousin the sea-otter, more decidedly aquatic and subsisting on shellfish and molluscs, has blunter molars recalling the teeth of the wolf-fish whose diet is similar. In the seals the forms of the cheek teeth are beginning to approach the homodont pattern and the distinction between premolars, molars and incisors is disappearing: in the fur-seals and sea-lions, revealed by their external ears, hairy fur and powers of land locomotion as being relatively recent recruits to the sea, this change has not gone far: but the common seals have regressed farther and the approach to a simple homodont dentition is unmistakable. In cetaceans, which for long have been so exclusively marine as to have lost their hind limbs, the change is complete and the dolphins and porpoises have teeth like fish. The whales which have given up a fish diet have gone even further and the whalebone whales subsisting on plankton need no teeth at all and have become edentulous (Fig. 6).

So much for the influence of dietetic function on form. But teeth sometimes serve other purposes—the tusks of the boar have already been mentioned: in some species of deer, hornless or with small horns, the upper canines usually absent in pecora are retained in the male as sexual weapons and in many primates such as the baboons and anthropoid apes the canines though present in both sexes are much larger in the male. The canine tusks of the walrus originally developed for combat are now employed for locomotion in hoisting the animal on to the ice. The incisors of rodents have been developed as tools with a wide range of employment in addition to their use in eating. To maintain a sharp cutting edge the enamel is thick in front and very thin or absent behind: the more rapid rate of wear on the posterior aspect ensures a chisel anterior edge and the continuous wear of the tooth is compensated by persistent growth (Fig. 3). Finally there remain some puzzling cases like the tusks of the elephants (originally the second upper incisors) and the upper tusks of the babirusa which are not anatomically in the mouth at all and which though they may have started their development for use in sexual warfare have progressed far beyond the optimum size for such a purpose and being of persistent growth appear to be more of a liability than an asset. In its evolutionary story the ever-increasing weight of the elephant's tusks led to a compensatory shortening of its neck: it was to reconcile this with its growth-in height that in miocene times the trunk came to be

developed. In these cases we see the obscure operation of an evolutionary momentum which seems to decree that an increase in size once started often progresses beyond the optimum and becomes a handicap to the species. The gigantic size of the mesozoic reptiles is an example, perhaps the brain of homo sapiens may prove to be another.

Are there characters in man's dentition compared with that of other anthropoids in which we can trace the influence of function ? No-one now believes he is at all closely related to living anthropoids and there is no reliable evidence that his ancestors had large canine teeth comparable with theirs. The Piltdown mandible is more and more generally believed to be unrelated to the cranium, and the form of man's milk dentition, always a pointer to the ancestral condition, does not suggest large canines there. But the adoption of the erect posture by man flexed the cranium on the vertebral column and led to a marked shortening of the body of the mandible to avoid pressure on the vital structures in the neck : crowding of the molar teeth was mitigated by a change in their shape, the mesio-distal becoming reduced somewhat by contrast with the bucco-lingual diameter if compared with the molars of other anthropoids. Another change in form can be correlated with the development of the hand. When the teeth of man are compared with those of the anthropoids the contrast in the size and strength of the front teeth is much more marked than in the case of molars. Now we know that the use of tools, hand-axes and scrapers, marks an early stage in man's evolution and one of their earliest uses must have been first to kill game or dig for food than to cut it up, in fact do the work previously done by the front teeth which, in accordance with the well known principle of evolutionary regression attendant on diminished use, began to shrink in size. The use of fire came much later in man's story than the use of flint, and it was not till fire made cooking possible that the cheek teeth enjoyed the easement felt long before by the front ones : this happened too late in time to have affected the size of the molars (Fig. 5).

Man's unique function of articulate speech cannot be claimed to have affected tooth form, but it was responsible for drastic changes in the modelling of the mandible and the shape of the dental arches designed to give the tongue freer play. The posterior width of the arch greatly increased and the lower mandibular margins became everted instead of inverted as in the apes. It was this eversion combined with the shrinking of the incisors which produced the chin, that characteristic human feature. Other changes in the upper jaws and the temporo-mandibular joint came at the same time. But these matters lie outside the scope of my theme which is the form of teeth, and their relation to function. Concluding with man, the story finishes close to its starting point in the primitive omnivorous pattern to which most early mammals conform.

SURGERY OF THE BILIARY SYSTEM

Lecture delivered at The Royal College of Surgeons of England

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by

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To COVER THE WHOLE subject of biliary obstruction in a single lecture would be impossible ; I therefore propose to restrict my remarks chiefly to the surgical aspects of jaundice, and more particularly to the surgery of stone in the common duct, since it is in this field that the most recent advances in biliary surgery have been made.

Jaundice

Jaundice or icterus is the yellow staining of the tissues of the body which results from an excess of bilirubin in the blood ; only the central nervous system, the tears, the saliva, and the milk escape. It is a symptom common to many diseases of several distinct systems. It is best demonstrated in the ocular conjunctiva, or on compression of the mucosa of the hard palate or lips by a glass slide.

Jaundice must be distinguished from other forms of yellow discoloration of the skin ; notably (1) the brown pigmentation of Addison's disease, which is most intense in the face, neck, and superior extremities, but which may discolour also the mucous membrane of mouth or vagina ; (2) the slaty pigmentation of hæmochromatosis ; (3) the brownish pigmentation of face, arms and axillæ which sometimes occurs in toxic goitre ; (4) the yellow discoloration produced when atebrine is taken as a malaria prophylactic and which is most intense round the mouth and in the hands and feet ; (5) the yellow complexion of carotenemia which is seen most typically in diabetics on a vegetable diet and which is due to a high chromolipoid content of the blood ; (6) the yellow discoloration of workmen in trinitro-toluene, which does not usually stain the conjunctiva and (7) aurantiasis of the skin shown by people who consume large numbers of oranges. (8) Picric acid, consumed by malingerers, may lead to yellow discoloration of the skin and sometimes it has produced as well a chemical necrosis of the liver and a true jaundice.

The *degree of jaundice* may be recorded either as the *icteric index*, or as the *bilirubin content* of the serum. The upper limit of the normal icteric index is five, that of the normal serum bilirubin 1 mg. per 100 ml. Clinical jaundice is not apparent until the serum bilirubin has increased to 3 mg. per 100 ml., and the icteric index to about 15. There is thus a stage of subclinical or latent jaundice which can be detected only by

these laboratory measures. Conversely, in recovery, visible pigmentation of the skin may persist after the serum bilirubin falls to normal; bilirubin accumulates in the serum more quickly than in other tissues, and it is cleared from it more quickly too. The serum bilirubin and the icteric index are most valuable (1) in the detection of subclinical jaundice, (2) in the assessment of the intensity of jaundice, and (3) in the day-to-day estimate of progress.

The van den Bergh Reaction

Three *varieties* of jaundice (McNee) are commonly recognised. In hæmolytic, pre-hepatic, or retention jaundice, the bilirubin of the serum has accumulated more rapidly than it can be excreted; it has not passed through the liver cells, and it gives only a delayed or an indirect van den Bergh reaction. In obstructive, regurgitation, or post-hepatic jaundice, the serum bilirubin has passed through the liver cells into the bile passages and has been reabsorbed from them, giving a positive direct van den Bergh reaction; this is the variety of jaundice with which we are most concerned to-day. In toxic, infective, or hepatic jaundice the liver function is depressed and inadequate to excrete bilirubin in normal amount, so that a proportion of the excess bilirubin has not passed through the liver cells; a proportion has passed through them however, but the mucosa of the bile ducts is œdematous, and there is some obstruction with reabsorption of bile-pigment, so that a biphasic van den Bergh reaction is obtained.

The van den Bergh reaction thus seems to offer a clear differentiation of the varieties of jaundice, but unfortunately the test is less dependable in clinical practice than it is on the blackboard. The chief anomalies are these; (1) In hæmolytic jaundice bilirubin may be excreted in the bile in such large quantities that it is deposited in the smaller ducts as bile thrombi or even in the larger ducts as pigment stones; above the bile thrombi the canaliculi dilate and secreted bilirubin may be reabsorbed, so that an obstructive element is superimposed upon the hæmolytic. (2) In infective hepatic jaundice (epidemic jaundice for example), catarrh of the bile-ducts may precede liver damage and the van den Bergh reaction may be initially direct. (3) In obstructive jaundice liver function ultimately suffers as a result of the obstruction, and a biphasic van den Bergh reaction may then be obtained.

Urobilinogen and Urobilin

The bilirubin which enters the intestine by way of the biliary tract is reduced to urobilinogen by bacterial action. This intestinal urobilinogen is now submitted to one of two processes; (1) some is converted to fæcal urobilin (stercobilin) by oxidation and is excreted in the fæces, but (2) a proportion is reabsorbed from the intestine, enters the portal blood, and is carried to the liver to be almost immediately re-excreted in the bile, provided it is not present in the portal blood in excessive amount, and provided liver function is unimpaired. A small amount of the urobilinogen

in the portal blood passes through the liver to the systemic circulation to be excreted by the kidney. If the urobilinogen content of the portal blood is high (from an excess of urobilinogen in the intestine), or if liver function is damaged, the proportion of the absorbed urobilinogen excreted by the kidney rises.

The urobilinogen and urobilin of fæces and urine may be estimated. (Watson, 1944) and their determination may be of assistance in deciding which variety of jaundice is present.

The *fæcal urobilinogen* is proportionate to the urobilin secreted by the liver ; normally 100-200 mg. per day are excreted in the fæces (Watson, 1942), but this rises to 600-2,000 mg. in hæmolytic jaundice, falls, sometimes to less than 5 mg. per day, in obstructive jaundice ; in toxic and infective jaundice it varies widely.

The *urinary urobilin*, normally 0·5 to 1·5 mg. per day, is greatly increased in hæmolytic jaundice (in which urobilin is absent from the urine) and in toxic jaundice, reduced usually to zero in complete biliary obstruction (when the urinary urobilin is high).

Liver Biopsy

In practice the differentiation of the three varieties of jaundice by the methods mentioned above may be exceedingly difficult. Sometimes the only early and certain criterion of distinction between toxic and obstructive jaundice, between infectious hepatitis, for example, and stone in the common duct, is liver biopsy (Dible, McMichael and Sherlock, 1943). Miss Sherlock (1945) employs the following technique :—The patient lies supine in bed with the right side as near the edge of the bed as possible, and slightly tilted to the right by a pillow under the left side of the hollow of the back. The right arm is placed behind the head. After local anæsthesia has been obtained in integuments, pleura, and diaphragm, a 15 cm. cannula of one mm. bore, with trocar in place, is passed through the ninth or tenth intercostal space in the middle or anterior axillary line. The patient breathes in, breathes out, and then holds his breath to keep the lung displaced upwards and the trocar is pushed through the diaphragm into the right lobe of the liver. The trocar is withdrawn when the instrument is half-an-inch inside the liver substance, and a cylinder of liver tissue is obtained for histological examination by pushing in the cannula a further 4-5 cm. A 20 ml. syringe is connected to the cannula and suction is maintained during withdrawal. The method is particularly valuable for differentiating obstructive from toxic jaundice during the first week or two of the disease, when the dilatation by bile of the central canaliculi of the lobules is clearly distinct if the jaundice is obstructive, clearly absent if it is toxic or infective.

The General Features of Obstructive Jaundice.

This is the variety of jaundice which is of the greatest interest to the surgeon.

Bile pigment, having passed through the liver cells, is reabsorbed from blocked bile channels to accumulate in the blood, which it endows with the direct response to the van den Bergh reaction, unless the function of the parenchyma is damaged, as it usually is after a time; a biphasic reaction may then be obtained. Not only does bile pigment accumulate in the blood, but the bile salt content rises too. Pruritus is a harrowing symptom of obstructive (as of infective) jaundice. Pruritus occurs only if bile salts as well as pigment are present in excess in the blood, but it is not proportionate to the concentration of bile salts and it may vary in severity, or disappear for a time, even though the jaundice increases progressively. The patient's reaction to pruritus by scratching is said to be responsible for the furunculosis which accompanies obstructive jaundice, and which is limited to the more easily accessible areas of the body surface; scratching is certainly responsible for the highly polished nails of the pruritic patient.

The bile salt content of the serum has other physiological and clinical effects than the production of pruritus. The surface tension of the blood is lessened, its viscosity is diminished, and its sedimentation-rate is accelerated. The bile-salts seem to be responsible too, by a depressant action on the central nervous system, for the mental depression, head-aches, drowsiness, irritability, bradycardia, and low systolic pressure which jaundiced patients present.

The coagulation time is increased in obstructive as in hepatic jaundice; the tendency to bleeding is greater in long-continued complete biliary obstruction than in any other form of jaundice. Not only is prothrombin formation impeded by liver damage (as in the toxic and infective types), but lack of bile-salts in the bowel prevents the absorption of the vitamin K which is necessary for the elaboration of prothrombin. The bleeding tendency in jaundice, and its control, is discussed later. It may induce purpuric eruptions and petechiæ.

The stool is colourless (clay stool) for so long as biliary obstruction is complete. It is bulky and offensive from an excess of split fat (steatorrhœa) —fat which has been hydrolysed to glycerol and fatty acid by pancreatic juice, but whose absorption is difficult in the face of shortage of bile salts. Constipation is invariable, with abdominal distension and anorexia, perhaps, also, from shortage of bile salts. The urobilinogen of the stool falls if complete biliary obstruction persists for long. Knowledge of the presence in or absence from the intestine of bile need not attend the passage of a stool; if a Ryle's tube is passed and if bile is withdrawn by it, it is clear that there is not a complete biliary obstruction present. The converse is not necessarily true. Bilirubin and bile salts both appear in the urine—sometimes before jaundice is clinically evident. The urobilinogen of the urine falls, but only if obstruction remains complete for days or weeks, as in malignant forms.

If biliary obstruction continues for long, liver function suffers, as function tests may show. In fatal cases the terminal phase is one of

complete liver failure or " cholæmia." There is restlessness or drowsiness, muscle tremors, twitching, convulsions, sweating, pyrexia, and, finally, a " typhoid state " with plucking at the bed-clothes, subsultus tendinum, and renal failure.

The two commonest forms of biliary obstruction are stone in the common duct and cancerous occlusion of the bile passages; the latter is dealt with by another lecturer.

Origin of Common Bile-Duct Stones

Stones which produce obstruction of the common duct may have arisen in the common duct, in the gall-bladder, or in the hepatic or intra-hepatic ducts. They may be of any composition to which gallstones are open. Infective stones have their origin usually in the gall-bladder, migrating to the common duct by way of the cystic duct while they are yet small, and growing in the common duct, or, much less commonly, ulcerating through from Hartmann's pouch to the common duct when of a larger size. Cholesterol stones, according to Walton (1937) always have their origin in the gall-bladder, never in the common duct, but there may be occasional exceptions to this rule, for Judd and Burden (1926) reported an intra-hepatic stone of pure cholesterol. The site of origin of pigment stones is frequently in the bile ducts, certainly in the smaller intra-hepatic ducts as bile thrombi, and probably also in the larger ducts. Stones in the ducts seldom form around liver flukes.

Whatever and wherever its origin, a stone lying in the common duct acquires a pultaceous crust of bilirubin-calcium and grows to an ovoid form. Above the stone, secondary stones may arise, or the duct may be distended with a mass of gravel or sand. In about 70 per cent. of cases the stone is single; in the remainder, from two to six stones are found, rarely more, though Mayo-Robson (1904) once counted 88, and Judd (1931) more than 100.

Situation of the Stone

In most cases the stone lies towards the lower end of the common duct, and some ten per cent. are found lodged in the ampulla (Judd and Marshall, 1930). Rarely are stones found in the hepatic ducts, or even within the liver (Lewisohn, 1916). Yet intra-hepatic stones are seldom solitary; they are usually accompanied by another stone, or other stones, at a lower level.

It should be remembered that a stone need not lie within the common duct to produce occlusion of its lumen; a stone in Hartmann's pouch may compress the adjacent common duct or produce œdema of its wall. Non-calculous cholecystitis sometimes exercises a similar effect.

The *diameter* of an obstructive stone may be as small as a few millimetres or as large as a few centimetres.

The Pathological Effects of Calculous Obstruction

A calculus may produce a continuous partial, or a recurrent ball-valve effect (Osler, 1897). A calculus which at first is firmly impacted with

complete occlusion may subsequently become loose. This is due to a widening of the ducts above the stone and extending down to the actual part of the duct in which the stone lies (Rolleston and McNee, 1929). Obstruction is complete in only 12 per cent. of cases (Watson, 1940).

When obstruction is present, there is moderate dilatation of the bile-passages above, whose walls are somewhat stiffened by inflammatory fibrosis. Ulceration is rather uncommon and perforation of the occluded duct is distinctly rare. When perforation does occur it is due to a combination of unfavourable factors (Newburgher, 1938) : increased pressure within the ducts, infection of the duct wall, thrombosis of adjacent arteries, and erosion by regurgitating pancreatic or duodenal secretions.

The *bile in the dilated duct* above a stone may be thick biliary mud or, if infected, frank pus. If, however, the content of an obstructed duct remains sterile, the bile pigment is reabsorbed from the duct's content. If liver function is so seriously affected that pigment excretion ceases, the content of the duct becomes clear, and increases by watery mucus from the glands in the duct wall (Fenger, 1896) ; this " white bile " is usually a sign of severe liver dysfunction (Rous and McMaster, 1921).

The *gall-bladder* remains contracted, in conformity with *Courvoisier's Law* (1890), in 80 per cent. of cases ; in these its dilatation is prevented by old-standing inflammatory fibrosis of the gall-bladder wall. In the remaining 20 per cent., in which the gall-bladder dilates as above a neoplastic obstruction, the occlusion is found to be due to a pigment stone which has formed within the ducts, or to a cholesterol stone from a gall-bladder, the seat of cholesterosis, but not grossly inflamed ; more rarely, the gall-bladder is dilated as a mucocele or empyema because the cystic duct is separately occluded by stone.

The *liver effects* are variable. In early operations, little abnormality is to be seen. If, as usually happens, the obstruction is associated with mild infection, the ducts within the liver show only slight dilatation, there is only a slight necrosis of liver cells, and some peri-ductal round-cell infiltration. If infection is severe and the bile-ducts filled with pus, dilatation is minimal, round-cell infiltration is widespread, and there may be miliary abscesses which sometimes fuse to form branching " cholangitic abscesses." Rarely, and with aseptic stone, the liver may show a pure hydrohepatosis as it does in malignant obstructions. Two further liver manifestations have been described as effects of stone— subacute liver atrophy (Snell, 1936) and chronic portal cirrhosis (Gibson and Robertson, 1939). I have seen the former of these at autopsy and have speculated whether the appearances might not be interpreted as infectious hepatitis occurring by chance in a patient who happened to have a stone in the common duct. When at operation I have observed the appearances of Laennec's portal cirrhosis in a jaundiced patient with a stone in the common duct, I have considered them to be due to the concomitant and

independent occurrence of stone and cirrhosis, diseases so common that it would be surprising if they were not sometimes found together.

Incidence

Walters and Snell (1940), quoting Judd's figures, state that stones are found in the common duct in 13 per cent. of all cases of biliary calculous disease ; in 6 per cent. the common duct is affected alone, and in 7 per cent. the gall-bladder too is the seat of stone. Other authors have found stones in the common duct in 13 (Allen, 1936) to 20 (Bernhard, 1935) per cent. of their cases of calculous biliary disease. Females are affected more often than males, but the sex ratio varies remarkably from author to author. Trueman (1940) found the ratio to be 3·2, Cutler and Zollinger (1940) 9.1 ; British figures approach the higher of these.

Clinical Features

The clinical picture was first described by Fernel (quoted by Major in his " Classic Descriptions of Disease ") but its cardinal features are summarized in Charcot's (1877) *intermittent hepatic fever*—intermittent pain, intermittent fever, intermittent jaundice, and loss of weight. Not all these symptoms, however, need be present together, and sometimes, perhaps in 20 per cent., the stone is entirely symptomless.

Pain is entirely absent in only a small number of patients with calculous jaundice—2-6 per cent. according to Jordan and Weir (1932). The jaundice is most likely to be painless if the gall-bladder is healthy and capable of dilatation, and it is just in these cases that a diagnosis of malignant obstruction may be made. Pain, if it is present, takes the form of a colic in 75 per cent. (Walters and Snell) ; in the remainder there is merely a lumbar or epigastric ache, or a rather painful form of gall-bladder dyspepsia, interrupted by rather more severe episodes. The pain may be located in the epigastrium or right hypochondrium or even in the lumbar, left hypochondriac, precordial, or infrascapular region. Typically, it is in the hypochondrium, radiating to the left side and to the back, but precordial pain may radiate down the left arm and " anginal attacks " have been precipitated by mechanical distension of the common bile duct (Ravdin, Royster and Saunders, 1942). Gilbert decreased the coronary flow in dogs by distension of the bile ducts.

Fever is present in only one-third of all cases. It usually takes the form of a daily rise to 100°—103° during an attack of colic, and is accompanied by a feeling of chill, but only in 4 per cent. by rigor. The presence even of high fever, does not necessarily mean a gross infection of the ducts ; even in Charcot's day, when prolonged intermittent fever with rigors was commoner than now, it was appreciated that Charcot's syndrome did not require a suppurative cholangitis for its production. Fever may occur without cholangitis and cholangitis without fever.

Jaundice of obstructive type may be considerable during complete obstruction, mild in incomplete obstruction ; it is absent in rather less

than a quarter of all cases—13 per cent. according to Jordan and Weir, 26 per cent. according to Judd and Marshall, and 35 per cent. according to Trueman. The icteric index is not so high as in malignant obstruction, seldom rising higher than 50, and it is usually subject to a great deal of fluctuation ; it seldom increases progressively over four successive days, while the jaundice, as measured by the icteric index, in malignant obstructions, though it may sometimes lessen and even disappear for a time, can always be shown to increase progressively over much longer periods. The serum bilirubin seldom exceeds 10 mg. per 100 ml. (Weir, 1932). Similarly, the stool is not usually completely colourless for longer than a few days ; bile pigment continues in the intestine, though in decreasing amounts, for a few days after biliary obstruction becomes complete, but the stool colours as soon as the duct opens. The laboratory findings are those of obstructive jaundice in general, but usually of an intermittent type, and sometimes complicated by those of a toxic jaundice, if liver damage supervenes.

Jaundice may be absent, of course, and other symptoms marked, if an external biliary fistula has persisted after a previous inadequate operation or if it develops spontaneously, as it rarely does. The establishment of an internal fistula with the duodenum, small intestine, stomach, or some other hollow organ, which occurs in 5 per cent. of cases, may have the same safety-valve effect, and is compatible with long life (Wakefield, Vickers and Walters, 1939).

On examination, the patient suffering from acute calculous obstruction of the common duct complains of tenderness in the epigastrium or right hypochondrium. The gall-bladder is not usually palpable. X-ray may show the presence of a stone or stones, but this does not necessarily mean that these lie in the common duct ; a stone lying in Hartmann's pouch may compress or inflame the adjacent common duct, obstructing it. Cholecystography should not be undertaken in a jaundiced patient ; it may exercise a severe toxic effect on a liver perhaps already damaged, and its administration in jaundice has occasionally been followed by acute pancreatitis.

The effect of previous cholecystectomy, or of co-existent obliterative gall-bladder disease, in a patient suffering from a stone in the common duct is usually to exaggerate the symptoms and to expedite their onset, for the dilator capacity of the gall-bladder endows it, to a limited extent, with the properties of a safety-valve for intraduct pressure. This property is lost largely if the gall-bladder is contracted by inflammatory fibrosis, and entirely if the gall-bladder has been previously removed (Mann and Bollman, 1924). If, however, a relatively normal gall-bladder is present, the onset of jaundice may be substantially delayed, sometimes beyond the time required for the stone to disimpact ; this may be the explanation of the slightness of symptoms in some cases of undoubted impaction of common duct stone.

The *course* of obstructive jaundice due to stone is usually self-limiting in any individual attack. Sooner or later the stone nearly always disimpacts, and obstruction seldom remains complete for more than a few days, but each episode of obstruction is an added insult to liver function. Very rarely spontaneous cure occurs by the development of a fistula between bile duct and duodenum or small intestine—natural choledochenterostomy.

Differential Diagnosis

The differential diagnosis of stone in the common duct from infectious hepatitis on the one hand, malignant biliary obstruction on the other, may be exceedingly difficult, for the first may be painless and apparently continuously progressive, the second may in its early stages be predominantly obstructive, and the third may be painful and apparently intermittent; the gall-bladder above an impacted stone may be palpable, that above a tumour may not be felt; the van den Bergh reaction may be biphasic in stone and tumour, direct in the infectious form. The whole picture may thus be confused, and the diagnostician dependent upon a majority rather than an unanimity of clinical and laboratory evidence, and compelled to delay his opinion for a few days. A really high icteric index and serum bilirubin, increasing on each successive day for more than four days is indicative of tumour; occult or frank blood in the stool or the duodenal content recovered by Ryle tube is suggestive of an ampullary tumour. Liver biopsy is of the greatest value in detecting or excluding infectious hepatitis. A history of previous jaundice many months before, or of typical gall-bladder dyspepsia of long duration, if either of these is elicited, is perhaps the most valuable single testimony in a case of jaundice, to the presence of a stone in the common duct. One of the most confusing forms of calculous jaundice is that which occurs when a pigment stone obstructs the common duct of a patient who has hæmolytic jaundice.

The Management of Calculous Biliary Obstruction

The management of calculous biliary obstruction is less generally conservative now than it was before the introduction of vitamin K. Even in 1939 Walton advised early operation to interrupt progressive liver dysfunction, and recorded a mortality of 14 per cent. in 197 cases. Until a few years ago a conservative management was imposed by the risk of hæmorrhage—Illingworth (1939) recorded 25 per cent. of post-operative hæmorrhages and 16 per cent. of fatalities from hæmorrhage after operations for stone performed in the presence of jaundice. Now it would be fair to say that, whether jaundice is present or not, the treatment of stone in the common duct should be preparation by carbohydrate diet and vitamin K (2 mg. intravenously) for two to three days, and, after that interval, operative removal of the stone or stones and drainage of the common duct. During the period of waiting the progress of the jaundice

is followed carefully. If during that period the degree of jaundice lessens clinically, if the icteric index and serum bilirubin fall, if bile decreases in the urine and reappears in the stool or in the duodenal content withdrawn by Ryle tube, and if urobilinogen increases in urine and fæces, operation may be further postponed until jaundice has disappeared, or ceased to improve. If, however, jaundice decreases at any time, and laboratory tests show an increase of obstruction, operation should be undertaken ; continued fever is another indication that operation should not be long postponed even if jaundice is decreasing.

If the gall-bladder is present and diseased it may usually be removed at the operation for exploration of the duct. If the condition of the patient is poor, however, and the liver function suspected to be poor either by function tests or by a long history of jaundice, it is sufficient to deal with the duct obstruction ; cholecystectomy is best postponed to a later date. Indeed, in a few cases of long duration and great severity, complicated by suppuration in the bile passages, the operator may well be content merely to drain the common duct and to postpone not only cholecystectomy, but even removal of the stone, to a later date.

The pain of common duct obstruction, which may be severe during the days of preparation, is not adequately relieved by morphine, which contracts the sphincter of Oddi. Pethidine is a better anodyne.

Hæmorrhage in Jaundice

Fraser in 1938 collected for the British Association of Surgeons a composite series of 4,000 operations performed by British surgeons upon the jaundiced. The fatality rate from hæmorrhage in the whole series was 14 per cent. Bleeding was commonest in longstanding cases of deep jaundice, in patients, for example, who had suffered for 4-5 months from complete occlusion of the common duct by cancer of the pancreas. Illingworth (1939) recorded a series of 50 cases of obstructive jaundice treated by operation ; 12 bled after operation, eight of them fatally. This risk has been virtually abolished by the introduction of menadione therapy.

In 1934 Dam deduced, from the spontaneous hæmorrhages suffered by chickens on a fat-deficient diet, the lack of a hypothetical " Koagulation vitamin." Four years later, with Glavind, he showed the beneficial effect upon these hæmorrhages of alfalfa grass. Quick meantime (1935) had shown that jaundiced patients suffer from a deficiency of prothrombin, and Butt and his colleagues in the Mayo clinic suggested (1938) that vitamin K be used as a pre-operative measure in jaundice.

Prothrombin, a pro-enzyme associated with the globulins, when exposed to the action of platelet thrombokinase in the presence of calcium, yields thrombin, and this in turn alters fibrinogen to fibrin. Prothrombin is synthetised in the liver provided vitamin K is present.

Vitamin K[1] occurs naturally in plant structures concerned with photosynthesis, and may be extracted from Alfalfa (McKee and others, 1939), spinach, sprouting oats, soya bean and from putrifying extracts of fishmeal, bran and casein. Vitamin K[2] obtained from sardine oil, and phthiocol (Anderson & Newman 1933, 1934) from the microbacterium tuberculosis are of much lower activity than natural vitamin K[1] (Thayer et al, 1939). All are compounds of naphthaquinone.

The vitamin K of man's natural diet is oil-soluble, and is only absorbed in the presence of bile salts. So also is the synthetic menadione (Kapilon Glaxo, Prokayvite B.D.H., and Gerophyl A.D.C.). Water salts of menadione are now available, however, and include the sulphonate (hykinone), the diacetate (prokayvite oral), the diphosphate (synkavit), and the bisulphate. These can be given intravenously, intramuscularly or orally in doses of 2 mg. Usually they are best given by all three methods, though the intramuscular preparations often give rise to aseptic inflammation, in jaundiced patients, at the sites of their injection. Liver synthesis of prothrombin imposes an interval of 24 hours before the maximum effect is obtained, so that operation should be delayed for at least this period.

Not all jaundiced patients have a low prothrombin concentration, and not all jaundiced patients bleed, but it seems fairly certain that those with deficient prothrombin are those subject to hæmorrhage. Prothrombin estimations by the Quick method are a fair index of the liability to hæmorrhage ; the normal human " prothrombin time " by this method is 18-22 seconds, but the " prothrombin index " is a good rough-test. Normal blood and patient's blood are compared ; to each is added thromboplasten (a saline extract of lung or rabbit brain), and the clotting times are observed. The prothrombin index is the ratio *normal control by* 100 : *unknown*. An index of less than 50 per cent. threatens hæmorrhage.

The effect of vitamin K on the prothrombin index is of value not only in treatment but in diagnosis. If the prothrombin index rises to the neighbourhood of 100 after twenty-four hours of vitamin K administration, the liver function may be regarded as unimpared. If the prothrombin index fails to rise after vitamin K, it is unlikely that the patient's jaundice is due to stone ; it is more probably infective.

The influence of anæsthesia upon the liver and upon bleeding must not be disregarded. A long anæsthetic, or an anæsthetic toxic to the liver such as chloroform or pentothal, may tip a wavering balance ; a prothrombin index measured and found satisfactory before operation may be reduced by anæsthetic depression of liver function sufficiently to occasion post-operative hæmorrhage.

Choledocholithotomy

Stones are best removed from the bile ducts under general inhalation anæsthesia supplemented by curare. Pentothal induction is as dangerous

to the liver as chloroform and is avoided. If spinal anæsthesia is employed it must either be induced to a high level or else supplemented by general anæsthesia during manipulation of the common duct; tension on the duct is transmitted to the diaphragm and pain is referred to the distribution of the fourth cervical segment.

The incision may be either right paramedian or (Kocher's) right subcostal according as the patient's costal angle is narrow or broad. The peritoneum once opened, the gall bladder is inspected, palpated, and retracted upwards and to the right by a forceps applied to its fundus. The pancreas is inspected for fat necrosis and palpated for the induration of pancreatitis, a not uncommon concomitant of impacted ampullary stone, even in the absence of clinical evidence. The left forefinger is then passed behind common duct and portal vein through the foramen of Winslow, and the common duct can be palpated between that finger and the thumb. Almost the whole length of the common duct is accessible to this form of palpation; even a stone in the ampulla can often be felt if the forefinger, hooked downwards in the lesser sac, and the thumb compressed against the second part of the duodenum, squeeze the pancreas forward from between them. This palpation may dislodge the stone upward. It is then fixed in the supraduodenal part of the duct by thumb and forefinger while packs are introduced, one into Morison's pouch, one displacing the small intestine downwards, and a third passed to the patient's left. The free edge of the lesser omentum is then balanced like a bridge on the forefinger lying behind it in the epiploic foramen of Winslow; if the stone lies, or has come to lie, at this level it is fixed on the finger by the anterior thumb.

The peritoneum is then divided carefully over the duct. This step is important. If the duct is blindly incised either the portal vein or an artery may be damaged, Particularly if the duct is narrowed by stricture, and the portal vein distended from hepatic venous obstruction, the vein may bulge round the right edge of the duct and lie partly in front of it. The arteries which may on occasion lie in front of the common duct, and be damaged if too boldly incised, are: (1) the right hepatic artery, twisting to the right in a " hump " in front of the duct; (2) the cystic artery, which passes in front of the common hepatic duct in 16 per cent. of cases, and in a considerable proportion of these, sometimes arising abnormally from the gastroduodenal artery (one half of one per cent.), crossing the common bile duct at a relatively low level; (3) an accessory cystic artery arising from the gastroduodenal ($5\frac{1}{2}$ per cent.) or superior pancreatico-duodenal (one half of one per cent.), ascending almost vertically on the front of the common duct; (4) the gastroduodenal artery itself, lying, as it sometimes does, in front of the common duct just above the duodenum. Injury of any of these vessels has a high nuisance value in an operation which should not be protracted, and ligation of the right hepatic artery in a patient whose liver function has already suffered, is extremely serious.

Even when the wall of the bile duct is clearly exposed, it may be difficult to be certain that it is not in fact the portal vein, especially if operations have been performed previously in the same area; it is legitimate and safe to aspirate content by syringe and fine hypodermic needle.

If the stone is fixed by the fingers at the required level—midway between upper border of duodenum and origin of cystic duct—an incision may be made down upon it. Otherwise two stay sutures are inserted, one near each border of the duct, and a vertical incision is made between them. Bile is aspirated as it escapes and its colour, important in prognosis, is observed; sometimes the first bile to escape is green, the colour gradually fading with successive escape until it is nearly clear; a specimen of the aspirated bile is retained for culture. A scoop of suitable size is then passed, first upwards and then downwards, and all stones are removed. The duct, if it contains mud, sand or gravel may be washed out with saline or with solution G. Forceps do not usually succeed when the scoop fails.

The duct is next explored for patency either with a flexible bullet-headed probe, or a Lister's bougie, or a scoop; whichever of these is employed it should be passed through the ampulla to the duodenum. In 80 per cent. of common duct obstructions I find the duct capable of exploration by the little finger.

This is the operation of *supraduodenal choledochotomy.*

In some cases a stone is so firmly lodged in the ampulla that it can be dislodged neither upwards nor into the duodenum. The operation of *transduodenal* choledochotomy is then performed. A longitudinal incision is made through the anterior wall of the duodenum, and the ampulla is inspected from within. If the stone is lodged in the ampulla, the edges of the ampulla are incised and the stone is removed. If the stone is lodged in the intramural portion of the common duct, the overlying duodenal wall is cut through to the stone. The anterior wall of the duodenum is then closed in two layers transversely. Drainage of the common duct is unnecessary after the transduodenal operation, but it is wise in these cases to perform supraduodenal choledochotomy as well, since only this operation allows adequate exploration of the duct for secondary stones.

The vast majority of stones may be removed by one or other of the operations described. A third technique, the *retroduodenal choledochotomy* of Kocher, is required only in the rare circumstance of a stone which has ulcerated through the infraduodenal segment of the duct wall to lie in a cosy diverticulum, or even to be extruded partly or wholly into periductal tissue. An incision is made around the upper angle of the duodenum, through the peritoneum of the posterior abdominal wall just beyond its reflexion from the duodenum, and through the fascia of Treitz, which thickens to form a sling support for the superior duodenal angle. A finger introduced through this incision into the retroperitoneum elevates forward the angle of the duodenum and the upper related part of the head of the

pancreas, and swings them medially to expose the infraduodenal portion of the common duct. This may be incised to free an incarcerated stone.

Drainage of the common bile duct is desirable after most if not all operations for the removal of stone. It may be omitted if a single stone is removed by transduodenal choledochostomy. It is essential if the duct contains pus, mud, sand or gravel, or if the bile content of the duct is colourless. The single hepatic drain of Mayo Robson (1894) may be passed well up into the hepatic duct, and anchored to the duct wall by a single catgut stitch. If the duct contents are purulent, it is wise to pass one tube upwards towards the liver, and a second downwards towards the ampulla (Moynihan 1926); there is no advantage in passing the downward-directed tube into the duodenum, as McArthur (1923) advised. These methods have now been largely replaced by the self-retaining T-tube of Kehr (1913), whose transverse limb, 4 cm. long, lies in the duct, and whose long vertical limb is brought out through the wound to drain into a bottle by the bed. Unfortunately there is a nine-months' delay in the supply of this tube in England to day, and its life is relatively short, so that resort must sometimes be had to the other drainage methods. Whichever method of drainage is employed, the opening in the duct may be closed round the tube or tubes by tying together the stay sutures previously inserted. If the opening is long one or two additional interrupted sutures of catgut may be added.

Post-Operative Care

Blood is usually required on the day of operation. The high carbohydrate diet and the administration of vitamin K, begun before operation, are continued after it. Daily specimens of bile are kept as a record of the progress of bile excretion, the behaviour of the icteric index or serum bilirubin is recorded, and the colour of stool and urine is estimated. The progress of the fæcal and urinary urobilinogen is also of interest— the latter often rises remarkably after operation when it has been at a low level before it.

Perhaps the best estimate of the recovery of liver function is the colour of the draining bile. Even though " white bile " is drained at first, if the colour changes to yellow, or better, green on the day after operation, recovery of liver function can be predicted. The longer the draining bile remains colourless the less likely is recovery of liver function to be complete. The urinary output and the blood urea are also important. When operation is performed in the face of severe liver damage, it may be followed by the so-called *hepato-renal syndrome* of Boyce and McFettridge (1935). This rare and terrible post-operative condition is characterised by a complete failure, from the time of operation, of the secretion both of bile and of urine. The patient shows no other abnormality for a few hours, or a day or two, and then lapses into the state of " cholæmia " already described, with in addition a rising blood-pressure and blood urea. I have seen this follow the relief of malignant jaundice on

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three occasions, but have not myself observed it after removal of a stone from the common duct. In one patient, the liver showed a hydro-hepatosis, but the kidney no abnormality; in the second the liver showed a hydro-hepatosis, and the kidney an acute cortical necrosis; in the third patient the liver showed a generalised lobular necrosis (the hepatic arteries were patent and undamaged) and the kidney an acute cortical necrosis. This last patient's systolic pressure did not fall below 115 from the beginning of the operation until shortly before death, so anoxia played no part, in her at least, in the development of the syndrome.

The drainage-tube may be removed when it loosens, usually after 9-10 days. The consequent biliary fistula usually heals in a few days. Persistence of the biliary fistula, or pain when it closes, may be due to a residual stone, to spasm of the controlling muscle of the ampulla, or to associated pancreatitis. It is in this respect that the T-tube is most useful. Its long external arm may be clamped after eight days; bile is then compelled to pass through the horizontal limb of the T into the distal part of the duct. If pain develops within six or eight hours of clamping the horizontal limb, draininge is re-established by removing the clamp. If no symptoms follow clamping of the tube it may be withdrawn.

The drainage tube, of whatever type, may also be used for cholangiography a week or so after operation. Opaque fluid, perabrodil for instance, injected along the tube, outlines the whole biliary tract and demonstrates the degree of dilatation of the ducts within the liver, the occurrence and extent of leakage from the common duct, and the presence or absence of obstruction at its lower end.

If bile leakage, continuing after removal of the tube, raises the suspicion of a residual stone, further operation should be deferred for at least six months. Spontaneous closure of a fistula due really to pancreatitis or to some unknown cause, may occur even after this period. The late Sir David Wilkie used to say that the ultimate criterion of high aptitude for a surgical career was not dexterity of hand or brilliance of intellect, but the ability to see weekly for six months a patient with a biliary fistula, and never to mention the word bile.

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ROYAL COLLEGE OF SURGEONS IN IRELAND.

RESUMPTION OF CHARTER DAY DINNER

CONFERRING OF HONORARY FELLOWSHIPS

THE TIME-HONOURED Charter Day Dinner of the College, traditionally held in February of each year, had been suspended during the war years. The last occasion on which the Dinner had been held was in February, 1939. The suggestion of the President, Mr. Frederick Gill, that the celebration should be resumed this year was welcomed by the Council, and the Dinner took place on February 14th, in the Large Hall of the College. Some 140 Fellows and their guests attended.

Before proceeding to the dining-hall, a pleasant ceremony was held. After the President had received the guests in the Council Room, the Honorary Fellowship of the College was conferred by him on Sir Alfred Webb-Johnson, Bt., K.C.V.O., D.S.O., President of the Royal College of Surgeons of England, who was presented for admission by the Secretary to the Council in the following terms :—

Mr. President : Little more than two hundred years ago, thanks in large part to the personal influence of his Irish surgeon, Louis XV of France lifted the craft of surgery from its lowly estate by his foundation of the Académie Royale de Chirurgie at Paris. That was in 1731. Thenceforward, the long-scorned surgeon could account himself a " man of no trade but of the profession of surgery only." Within half a century, Edinburgh and Dublin had followed suit, and, under Royal Charter of February 11th, 1784, the anniversary of which grant we meet to celebrate this evening, the Royal College of Surgeons in Ireland was born, not unfittingly, in the Rotunda Hospital.

Shortly after their incorporation, the Members of this College decided to elect men of distinction in the craft from other shores as Honorary Members of their College. Most properly to-night we recall that the first name inscribed on our Honorary Roll is that of Robert Adair, one-time surgeon to Chelsea Hospital, famous in his day as " a favourite of princes, of women and of Fortune." To-night, Sir, it is my privilege to present to you for enrolment as our youngest Fellow, Sir Alfred Webb-Johnson, President of the Royal College of Surgeons of England. As surgeon to Chelsea Hospital, he is in the direct line of succession to our oldest Fellow, Adair, and to his greater successor at Chelsea, John Hunter, " the Thunderbolt of Surgery," whose name was entered on our Roll in 1790.

Sir Alfred's career has shown a curious parallel with that of the " fortunate Irishman," Adair. Like him, Sir Alfred has seen active service in Flanders fields, winning there high distinction for his work as an army surgeon, and earning thereby the favour and the confidence of Royalty ; later, in a second campaign, he has served as first surgical consultant to the War Office.

Another name here comes to mind. Like that great little man, Samuel Pepys, who though small of stature was stout of heart, Sir Alfred has seen his London

burn. Of true Pepysian mettle, he, too, disdained to leave the burning city, staying to watch over the office and its priceless treasures entrusted to his care. His Presidency of the College constitutes in itself a record : for no President heretofore has held that high trust for seven unbroken years.

Throughout those years, years of frequent anguish and well-nigh of ship-wreck, his has been the leading spirit in ensuring that his College, sadly stricken as it was, should again arise, Phœnix-like, from its ashes, a stronger and a greater Academy of Surgery than it had been before. Some had uttered the reproach that the College did no teaching : even through the stress of war, as President, he has seen to the establishment of three new Chairs, of Anatomy, Physiology and Pathology ; he has reanimated the College as a new centre of post-graduate teaching, and has taken care for its permanent educational endowment. By the example of his own generosity, by his enthusiasm, by his powers of per-suasion, he has ensured that the new College, rebuilt on its old site, shall be a temple of supreme surgical endeavour.

In this, his vision has been restricted by no narrow spirit of metropolitan ascendancy : rather has it been wide as the Seven Seas, including in its scope Egypt, India, Australia and New Zealand, and thereby co-ordinating the standards of surgical efficiency in those far teaching centres.

To Alfred Webb-Johnson, surgery, once a lowly trade, is to-day a high and holy calling, one knowing no boundaries of sect or school. He has set all who profess it the example of faithful and flawless service, of devotion to duty and of unselfish accomplishment. In honouring him, Sir, you do honour to us, his fellow-craftsmen, wherefore we now pray you to admit him, in company with Adair and Hunter, his ancestors of Chelsea, as an Honorary Fellow of the Royal College of Surgeons in Ireland.

The President then admitted Sir Alfred to the Fellowship of the College, and the newly elected Fellow signed the Roll.

Mr. Gill then declared that he would vacate the President's Chair to enable Sir Alfred, as President of the English College, formally to confer on Mr. Edward Sheridan, a past President of the R.C.S.I., the Honorary Fellowship in Dental Surgery of the Royal College of Surgeons of England.

Having thanked the President and Council for his election as a " new boy," Sir Alfred then spoke as follows :—

I am very grateful to you, Mr. President, for allowing me, your youngest Fellow, to greet Mr. Sheridan, your immediate predecessor in the Chair, and to admit him to the Fellowship in Dental Surgery recently instituted by the Royal College of Surgeons of England.

Mr. Sheridan, I count it a great privilege to have been entrusted with the pleasant duty of admitting you as a Fellow in Dental Surgery of my College. You have a remarkable record of achievement, for besides being an acknowledged leader in your branch of surgery, you have won the esteem and confidence of all who have been associated with you in your work. It will only fall to the lot of one of the very elect to be chosen successively by the Lord Lieutenant, then by his fellow dental surgeons, and then by the Privy Council, to sit as a member of the governing body of his profession, and finally to be selected to preside over it.

By kind permission of The Irish Times

Sir Alfred Webb-Johnson, Mr. William Doolin, and Mr. Frederick Gill, President of the Royal College of Surgeons in Ireland.

To have obtained such outstanding eminence in your special branch of surgery and at the same time to have won the approval of your general surgical colleagues, as shown by your election as President of this College, must be an absolutely unique record.

Is it any wonder, therefore, that the Board of the Faculty of Dental Surgery and the Council of my College felt that there would be a serious gap in the list of Fellows if your name were not included ?

In offering this recognition of your work and character, we realise that the inclusion of your name in the initial list of Fellows adds distinction to the Roll, and we trust that you will always feel that you have an academical and a spiritual home in Lincoln's Inn Fields. In the name, then, of the College, and by authority of the Council, I admit you as a Fellow in Dental Surgery of the Royal College of Surgeons of England.

Mr. Sheridan suitably replied.

After dinner, the toast of " Our Guests," was proposed by the Vice-President, Mr. H. S. Meade, and was replied to by the new Fellow and the President of University College, Dublin, Mr. Michael Tierney. The toast of " The College " was proposed by Professor J. W. Bigger, and was responded to by the President, Mr. F. Gill. The musical programme was in the capable hands of Dr. George Hewson.

We are indebted to Mr. William Doolin, F.R.C.S.I., Editor of The Irish Journal of Medical Science, for this report.

THE RESTORATION AND DEVELOPMENT FUND

THE AMOUNT RECEIVED or promised for the Restoration and Development Fund has now reached over £186,000. This is very gratifying when it is realised that there is not yet an atmosphere of urgency, for it is not possible to proceed with building operations in the present state of the country and the acute shortage of labour and building materials. Extensions into neighbouring houses are however being made, and it is hoped in the near future to open a centre for post-graduate students, providing a bureau, collegiate amenities such as common rooms, &c., and a limited amount of residential accommodation.

In addition to the gifts recorded in the Annals of July last year, some notable sums have been received since the 1947 Anniversary of Hunter's birth. Viscount Leverhulme has made the generous gift of £1,000 to mark his election as a Trustee of the Hunterian Collection. The Johnson and Johnson Research Foundation made a handsome gift of £1,000, and this has been followed by a further donation of £500 from Messrs. Johnson and Johnson (Great Britain) Ltd. This lead has been followed by Messrs. Robinson & Sons and Messrs. Southalls, who have each given £100. It is hoped that other surgical instrument and dressings manufacturers will also contribute.

Mr. Lawrence Abel has given five hundred guineas and Mr. Sydney Gordon Davidson of Aberdeen, two hundred guineas.

Professor Evarts Graham, after his Lister Oration, made a gift of £175, and Dr. Frank Lahey gave one hundred guineas after delivering the Joll Memorial Lecture. Mrs. George Crile, widow of the world-famous surgeon, sent a spontaneous gift of five hundred dollars.

One hundred guineas has been contributed by each of the following: M. H. Phillips, Lt.-Col. H. G. Alexander, Robert Ollerenshaw, A. Harvard Baker, J. Rudd Ratcliffe, E. G. Muir, Miss E. L. Willis, Miss Dorothy Collier, The Practitioner, J. M. P. Clark, J. S. Batchelor, J. Duncan Gray, R. S. de Bruyn, Miss Edith May Hall, W. S. Fenwick, C. H. Medlock, R. C. MacQueen, P. G. McEvedy, R. A. Brews, Sir Chad Woodward, R. V. Cooke, Surgeon Captain W. J. Colborne, T. Ivor Hughes, A. M. Boyd, H. W. L. Molesworth, Alfred Hobson and A. R. Lister.

One hundred pounds has been contributed by Sir Lenthal Cheatle, A. L. Robinson, L'Assoc. Francaise de Chirurgie and Prof. Ian Aird.

Seventy pounds by H. E. Harding, W. I. Daggett, H. Jackson Burrows.

Fifty guineas by Maurice Backwell, A. J. B. Goldsmith, S. S. Beare, J. M. Fitton, C. K. Vartan, G. C. Sawyer, S. W. Maslen Jones, C. A. Lupton, G. M. FitzGibbon, W. E. Tucker and R. C. Davenport.

Fifty pounds by James Christie Anderson, Miss Crookshank, Lt.-Col. W. A. D. Drummond and R. N. Grant (a graduate student).

Many smaller donations have been received, but all gifts cannot be recorded here. They are none the less appreciated, for in a campaign like this it is the number of supporters that count as well as the amounts of the gifts, and the Fellows of the College have made a splendid response.

It is a most inspiring task to prepare plans for the future with so much loyal support.

4. THE CHESELDEN CUP

THE CHESELDEN CUP is a beautiful example of English plate of the Queen Anne period. It is a double-handled silver-gilt cup with cover, and was presented to the College by Mrs. Cecil Joll, whose husband was a great expert on old English silver. Cecil Joll bought the cup at Christie's on its merits and gave it to his wife. When he found, on inquiring at the College of Arms, that the Coat of Arms engraved on the cup was that of William Cheselden he persuaded Mrs. Joll to give it to the College, where it is one of the most prized possessions—not only for its intrinsic value and the fact that it belonged to Cheselden—but also because of the donor and the history of the gift.

William Cheselden (1688-1752) was a versatile man, famous as anatomist, surgeon and brilliant operator, and, as the first to perform iridectomy, a pioneer in ophthalmic surgery. This last activity is on record in Pope's " Imitations of Horace " : –

> " Late as it is, I put myself to School,
> And feel some comfort, not to be a fool.
> Weak though I am of limb, and short of sight,
> Far from a lynx, and not a giant quite ;
> I'll do what Mead and Cheselden advise,
> To keep these limbs, and to preserve these eyes.
> Not to go back, is somewhat to advance,
> And men must walk at least before they dance."

Cheselden was Lecturer in Anatomy, and Surgeon to St. Thomas's Hospital (1718-1738). He was elected F.R.S. in 1711. He was Surgeon to Queen Caroline, 1727, was elected Surgeon to St. George's Hospital on its foundation in 1733, and Surgeon to the Royal Hospital, Chelsea in 1737. He was Junior Warden of the Company of Barbers and Surgeons, 1744, Senior Warden of the new Company of Surgeons in 1745 and was elected Master in 1746 in succession to John Ranby, the first Master.

Cheselden was the first Corresponding Member of the Académie Royale de Chirurgie de Paris. He was chosen Sheriff of London in 1743 but was allowed to " swear off " on the ground that his estate in lands and goods was not of the value of £1,500. It is said that he drew the plans of the old Putney Bridge.

Cheselden was a pioneer in the teaching of anatomy outside the Hall of the Company of Barbers and Surgeons, and the activities of Cheselden and others led the way to the Hospitals and Medical Schools gradually assuming the functions of the Company in medical education.

The Cheselden Cup

Coat of Arms of William Cheselden

By the time of William and Mary, teaching was being carried on systematically at the two hospitals then existing in London—St. Thomas's and St. Bartholomew's. The governors of St. Thomas's recognised the right of the surgeons to take pupils, but advised that " none shall have more than 3 cubbs at one time." The Company of Barbers and Surgeons, however, had exclusive rights in regard to teaching, and complaints were made " against breeding soe many illiterate and unskilful pretenders to Chyrurgery at St. Thomas's Hospitall." At a later period the Company took disciplinary action against those who taught anatomy and dissected in their own houses, and Cheselden was among those called to account. The opposition of the Company to these developments provides an interesting manifestation of the unthinking defence of exclusive rights, for surgery was being established on a sure scientific basis by the surgeons who were teaching in their hospitals and in their private classes.

It is interesting to note that the Royal College of Surgeons is now resuming regular teaching activities in Surgery and the allied Specialties, and in the basic medical sciences of Anatomy, Pathology and Applied Physiology—but in a more fitting capacity, confining its activities to post-graduate students.

Other mementos of William Cheselden at the College besides the Cheselden Cup are a terra-cotta bust by Roubillac, the well-known portrait by Jonathan Richardson, and the Cheselden Medal (a prize awarded at St. Thomas's Hospital) by W. Wyon R.A., one of the most famous of British Medallists.

A.W-J.

SAYINGS OF THE GREAT

" No sadder proof can be given by a man of his own littleness than disbelief in great men."—*Carlyle.*

" Don't think, try the experiment."—*John Hunter.*

" *Dei laboribus omnia vendunt.*"—" For toil the Gods sell everything."—*Harvey.*

" A man should never be ashamed to own he has been in the wrong, which is but saying that he is wiser to-day than he was yesterday."—*Pope.*

" God made all men equal is a fine-sounding phrase and has also done good service in its day, but it is not a scientific fact."—*Winwood Reade.*

" Drink no longer water, but use a little wine for thy stomach's sake . . ."—*1st Timothy, v.* 23.

DIARY FOR APRIL (15th—30th)

Thur. 15	3.45	PROF. H. A. HARRIS—Clinical Anatomy of the Chest.
	5.00	DR. G. POPJAK—Isotopes—Their Biological and Medical Application.
	6.15	DR. JOHN BEARD—Post-anæsthetic Treatment.
Fri. 16		D.M.R.D. and D.M.R.T. Examinations (Part I) and D. Phys. Med. Examination (Part II) begin.
	3.45	PROF. H. A. HARRIS—The Anatomy of Posture.
	5.00	PROF. M. J. STEWART—The Pathology of Peptic Ulceration.
	6.15	DR. WILLIAM W. MUSHIN—Applied Anatomy.
Mon. 19	3.45	PROF. LE GROS CLARK—The Optic Pathways.
	5.00	PROF. D. S. RUSSELL—Intracranial and Intraspinal Tumours.
	6.15	PROF. D. T. HARRIS—Physiology of Respiration.
Tues. 20	3.45	PROF. J. D. BOYD—Derivatives of the Pharyngeal Arches.
	5.00	PROF. D. S. RUSSELL—Intracranial and Intraspinal Tumours.
	6.15	DR. KATHERINE LLOYD-WILLIAMS—Anæsthesia in Obstetrics.
Wed. 21	3.45	DR. G. POPJAK—Isotopes—Their Biological and Medical Application.
	5.00	DR. T. A. SCHWEITZER—The Reflex Control of Blood Pressure and Heart Rate.
Thur. 22		L.D.S. Examination (Properties of Dental Materials) begins.
	3.45	PROF. LAMBERT ROGERS—The Cerebral Circulation.
	5.00	DR. T. A. SCHWEITZER—Measurements of Cardiac Output and Factors Influencing it.
Fri. 23		D.M.R.D. and D.M.R.T. (Part II) and Primary F.D.S. Examinations begin.
		Voting papers for Council Election issued.
	3.45	DR. F. C. COURTICE—Control of Respiration.
	5.00	PROF. J. H. BIGGART—The Pathology of Head Injuries.
Mon. 26		L.D.S. Examination (Dental Mechanics) begins.
	3.45	DR. F. C. COURTICE—Transport of Oxygen and Carbon Dioxide.
	5.00	PROF. J. H. BIGGART—Some Modern Views on Vascular Disease.
	6.15	SIR GORDON GORDON-TAYLOR—Glands in the Neck.
Tues. 27	3.45	PROF. S. ZUCKERMAN—Male Reproductive Organs.
	5.00	DR. C. R. HARINGTON—Thyroid Physiology.
	6.15	MR. R. MOWLEM—Injuries of the Nose and Sinuses.
Wed. 28	4.30	Meeting of Fellows.
Thur. 29	3.15	DR. F. S. GORRILL—The Lymphatic System.
	5.00	DR. M. G. EGGLETON—Modern Methods of Assessing Renal Function.
	6.15	MR. F. W. WATKYN-THOMAS—Tinnitus.
Fri. 30		Final F.D.S. Examination begins.
	3.45	MR. R. J. LAST—Segmentation of Limbs.
	5.00	DR. M. G. EGGLETON—Some Factors affecting Renal Function in Health and Disease.
	6.15	MR. R. G. MACBETH—Osteomyelitis Secondary to Sinusitis.

DIARY FOR MAY

Mon. 3		Final F.D.S. Oral Examination begins.
	3.45	PROF. J. M. YOFFEY—Anatomy of C.S.F. Production and Absorption.
	5.00	DR. H. HELLER—Control of Water Metabolism by Pituitary Hormones.
	6.15	MR. I. G. ROBIN—The Treatment of Intrinsic Carcinoma of the Larynx.
Tues. 4	3.45	DR. H. HELLER—Control of Salt Metabolism by Adrenal Hormones.
	5.00	PROF. M. L. ESNAURRIZAR—The Surgery of Pain and Splanchnit Dystrophias.
	6.15	MR. D. F. A. NEILSON—Diverticula of the Pharynx.
Wed. 5	3.45	DR. J. WHILLIS—The Tongue and Soft Palate.
	5.00	PROF. R. HARE—The Source and Transmission of Nasopharyngeal Infection.
	6.15	MR. R. J. CANN—Meningitis in Diseases of the Ear and Nose.

Thur.	6	3.45 Dr. J. Whillis—Action of Muscles in the Forearm and Hand.
		5.00 Prof. R. Hare—The Source and Transmission of Wound Infection.
		6.15 Mr. W. I. Daggett—The Operative Treatment of Chronic Infection.
Fri.	7	3.45 Dr. W. F. Harper—Thyroid Gland and Pretracheal Region.
		5.00 Dr. C. C. N. Vass—Digestion.
		6.15 Mr. R. R. Simpson—Nerve Deafness.
Mon.	10	3.45 Dr. C. C. N. Vass—Digestion.
		5.00 Mrs. F. M. Bonser—Cancer of the Breast : the Use of the Experimental Method in the Elucidation of the Cause.
		6.15 Dr. I. Simpson Hall—Simple and Malignant Growths of the Ear.
Tues.	11	3.45 Dr. E. F. Gale—Principles of Chemotherapeutic Action.
		5.00 Mrs. G. M. Bonser—The Relation between the Factors Concerned in Carcinogenesis of the Breast and of Other Organs.
		6.15 Mr. G. Ewart Martin—Foreign Bodies in the Air Passages.
Wed.	12	3.45 Dr. E. F. Gale—Principles of Chemotherapeutic Action.
		6.15 Dr. H. A. Lucas—Nasal Allergy.
		7.00 Monthly Dinner for Fellows, Members and Licentiates.
Thur.	13	Final F.R.C.S. Examination (Ophthalmology and Otolaryngology) begins.
		3.45 Dr. C. H. Tonge—The Vertebral Column and its Movements.
		5.00 Prof. J. H. Dible—Inflammation and Repair.
Fri.	14	3.45 Dr. S. J. Folley—Modern Aspects of Lactational Physiology.
		5.00 Prof. J. H. Dible—Inflammation and Repair.
Mon.	17	College closed.
Tues.	18	3.45 Dr. G. L. Brown—Neuro-muscular Transmission.
		5.00 Mrs. E. K. Dawson—Carcinoma of the Breast.
Wed.	19	Final Fellowship Oral Examination (Ophthalmology and Otolaryngology) begins.
		3.45 Dr. G. L. Brown—Neuro-muscular Transmission.
		5.00 Mrs. E. K. Dawson—Sarcoma of the Breast.
Thur.	20	Final F.R.C.S. Examination (General Surgery) begins.
		3.45 Prof. J. H. Gaddum—Drugs Allied to Adrenaline.
		5.00 Dr. Cuthbert Dukes—Tumours of the Rectum and Colon.
Fri.	21	D.A. Examination begins.
		3.45 Dr. Cuthbert Dukes—The Kidney, Bladder and Prostate.
		5.00 Prof. J. H. Gaddum—Drugs Allied to Choline.
Mon.	24	Final Fellowship Oral Examination begins.
		3.45 Prof. T. Nicol—Selected Features in the Anatomy of the Pelvis and Perineum.
		5.00 Dr. J. R. M. Innes—The Comparative Pathology of the Central Nervous System.
Tues.	25	3.45 Prof. F. Wood Jones—Visceral Outlets of Hind End.
Wed.	26	5.00 Prof. A. C. Lendrum—The Surgical Significance of some so-called Simple Tumours.
Thur.	27	5.00 Prof. A. C. Lendrum—The Surgical Significance of some so-called Simple Tumours.
Mon.	31	3.45 Prof. F. C. Happold—Growth Factors.

SCROTAL SWELLINGS

SEVERAL MILLION YEARS AGO Nature decided that the creatures with which she had peopled the globe were getting out of date. They were large and looked terrific, but they were at the mercy of the temperature of their surroundings: when the sun shone they pranced, when the cold weather came they went to sleep. Something more efficient was needed, a creature designed to work at a particular temperature, and maintaining that temperature regardless of its surroundings. Among the many mechanisms that were introduced to put the new model on the market were a super-charger, the diaphragm, a better fuel pump, the four-chambered heart, high-octane fuel obtained by scrapping the nuclei of the red cells, shortening their life but making them better carriers of oxygen, and a really efficient radiator thermostatically controlled by the sympathetic system. One function alone refused to be redesigned, that of reproduction. It is remarkable how the faculty of reproduction can be carried out only under certain climatic conditions, and how to satisfy those conditions the life force impels creatures to face dangers and undertake feats that they would never contemplate in their own interests. Eels leave their ponds, and undertake a journey of two thousand miles over land and water to breed in the depths of the Saragossa Sea. Salmon come half across the world to a certain Scottish loch, and batter themselves against the rocks as they try again and again to leap the falls that intervene. Seals converge from the whole Pacific on the Aleutian Islands. Birds fly from South Africa to reach the cooler climate of Norway. The reproductive cells, at any rate the male reproductive cells, demand a lower temperature than that at which the other mechanisms work best, and the mammal has therefore been obliged to extrude them from the abdominal cavity to which they belong. Hence the scrotum.

Some mammals, chiefly aquatic, keep their testicles in the abdomen all the time. A few extrude them during the breeding season only. The great majority keep them in a scrotum, whose purpose seems to be to maintain them at a lower temperature than that of the rest of the body. In warm weather the scrotum relaxes; in cold weather it contracts, bringing its contained testicles close to the warm trunk. In animals equipped with a scrotum, warming the testis by an electric circuit, by wrapping the

scrotum in wool, or by returning the testis to the abdomen, renders a previously fertile male sterile. The dartos muscle, the hair, and the abundant sweat and sebaceous glands of the scrotum all seem designed to help its purpose in maintaining the testis at a lower temperature than the rest of the body.

The methods of clinical examination in all parts of the body are the same : inspection, palpation, auscultation and percussion, but the last two are of no particular value in examining scrotal swellings. In them, however, transillumination, a subvariety of inspection, becomes particularly important.

Inspection allows us to judge of the size, shape and symmetry of the scrotum, and thus to make some inferences about the normality or otherwise of its contents. Above all it allows us to recognise diseases of the scrotum itself which, however, are not usually included in the heading of scrotal swellings. Two may be mentioned : scrotal cancer, which, when it occurs in *sweeps and mulespinners, is classed as an industrial disease* and is certifiable, but which is also found occasionally in other workers in oil, such as garage hands, and filarial Elephantiasis, in which the scrotum may attain colossal proportions and may even exceed in weight the rest of the patient. We seldom see such trophies, but our colleagues overseas meet them frequently. Major F. A. B. Sheppard, late of Madras, writes to me : " In 1936 a clanking and rattling in the corridor outside my office brought me to the door. There, outside, about to enter was a thin middle-aged Indian supporting his enormous elephantoid scrotum on an old tin wheel-barrow which was much the worse for wear and collapsing under the weight. It was the dilapidated state of the wheel-barrow and the impossibility of prolonging its usefulness by further repairs that brought the individual for treatment. The scrotum when removed weighed some 75 lb. It was my custom to collect old coins and other objects removed from the body as souvenirs but I let the wheel-barrow go !

There was another, much older gentleman of 75, who reminded me of the character in Shaw's " Androcles and the Lion "—(his name eludes me)—who, realising that the end of his immoral life was near, decided to profess Christianity and become a martyr. The aged one of 75 had also led a similar life and decided for similar reasons to spend his remaining days in pilgrimages to temples. Unfortunately, his enormous scrotum more than got in his way—it completely immobilized him. He, therefore, decided it was time to have it removed. This one weighed only 55 lb., but in removing it I included also his testicles, thereby ensuring his better behaviour and probably a better place in a better world ! "

Palpation allows us to determine the shape, position and *consistency* of the scrotal contents. The normal scrotum contains only those structures extruded from the abdomen that it was designed to receive, that is, the

cord, testis and epididymis. The examiner sits facing the standing patient and feels the neck of the scrotum on the right side with his left hand, that on the left with his right hand. The index finger is passed behind the scrotum, and the thumb in front is slid outwards, rolling the structures of the cord between the two. The cord will be felt as a loose skein of irregular soft strands running longitudinally, the fibres of the cremaster muscle and the veins of the pampiniform plexus, among which the vas can easily be distinguished as a firm tube that used to be likened to whipcord, but that a generation who have never driven a horse nor cracked a whip would probably compare in feel and diameter to a ureteric catheter.

The cord leads down to the testicle. The words " testicle " and " testis " are variously used, limited by some to the body of the testis, employed by others to describe everything that hangs from the cord. Some use " testis " in the particular and " testicle " in the general sense. The inaccuracy is unfortunate, but to-day irremediable. The surgeon who is describing testicular swellings should specify whether he is speaking of the coverings, the body of the testis, or the epididymis ; the candidate, on the other hand, who is asked to discuss " testicular swellings " should describe those of the tunica vaginalis and the epididymis as well as the body.

The abnormal structures that may be found in a scrotum are swellings of the constituents of the cord, swellings of the epididymis, swellings of the body of the testis, swellings of the tunica vaginalis, and, in addition, swellings connected with the funicular process, the peritoneal pouch that · precedes the other structures into the scrotum and, having seen them safely settled in, usually disappears. The first point in examination, therefore, is to feel the neck of the scrotum and determine whether a swelling is inguino-scrotal or purely scrotal ; if it is inguino-scrotal it is almost certain to be connected with the funicular process, if it is scrotal it is almost certain to be connected with the testis complex. Therefore, having examined the cord as it lies in the neck of the scrotum, we next feel for the testis. If we can find it we examine separately the body and the epididymis. If we cannot identify it, the body of the testis and epididymis must either be hidden in a tunica vaginalis distended with fluid or with blood, or they must be so distorted by disease, which usually arises in the body, as to be unrecognizable.

If the swelling is inguino-scrotal it is almost certain to be a varicocele, or an oblique inguinal hernia, either of which can be recognised by any student within a week or two of leaving the dissecting room (Fig. 1). The only inguino-scrotal swellings likely to trap the experienced but unwary surgeon are a congenital infantile or bilocular hydrocele and a hydrocele in a hernial sac (Fig. 2). All of these present as hydroceles, but palpation of the neck of the scrotum shows that the swelling extends up into the inguinal canal. The only difference between them is that in the

case of the hydroceles the inguinal part of the swelling is fluid and can be felt to fluctuate in relation to the scrotal part, whereas the inguinal part of an enlarged hernial sac contains omentum, and is solid.
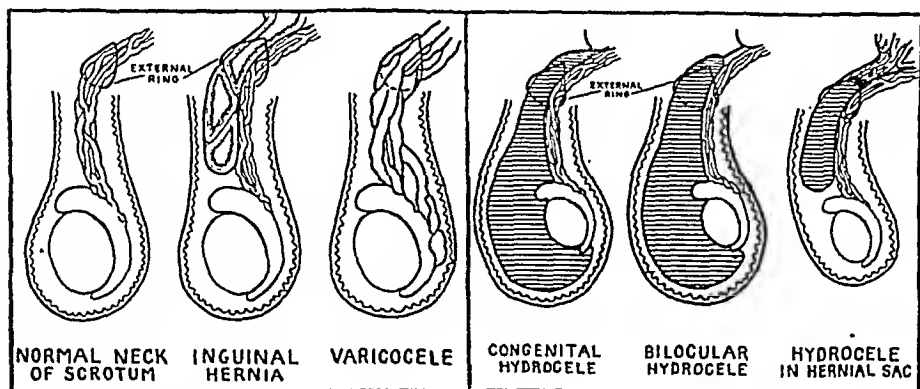

Fig. 1.   Fig. 2.

Rare swellings of the cord, which one seldom encounters, but which must be borne in mind are :—

1. *Diffuse hydrocele or multilocular cystic disease of the cord.* The funicular process, instead of undergoing obliteration, has remained as a chain of irregular serous cysts.

2. *Encysted hydrocele of the cord.* The funicular process has been closed at both ends but remains patent in the middle, and this sequestered section has become distended with fluid.

3. *Hæmatoma of the cord.* Ten years ago I received a message that the 15-year-old son of a great friend had strangulated an inguinal hernia at his public school, and was on his way to London by ambulance. The boy had been playing rugby football, when he had been seized with severe pain in the left groin and vomited. The doctor who was called found a tense, tender and irreducible swelling extending through the inguinal canal and along the cord as far as the testicle. Time, the solver of all conundrums, had made the diagnosis entirely clear by the time he reached me. He was now feeling well, and had a normal pulse. The sausage-shaped swelling in his scrotum had assumed a blue colour and was already staining the skin of the neck of the scrotum and it was clearly the result of the rupture of a vein of the pampiniform plexus inside the sheath of the internal spermatic fascia.

4. *Metastases in the lymphatics of the cord.* The primary tumour is usually testicular, but, as in a recent case under my care, it may be in the abdomen.

5. Rarest of all are innocent tumours of the cord, myoma or lipoma.

If the cord at the neck of the scrotum is found to be normal, the testicle is next palpated. When neither testis nor epididymis can be felt, but in their place there is an ovoid swelling, the cause is almost certainly distension of the tunica vaginalis, usually with clear fluid, occasionally with blood. Does it fluctuate? Then it contains fluid. Does it trans-illuminate? Then the fluid is clear. If it transilluminates, where is the testis?

Hydroceles fall into two groups, the secondary which are the result of neighbouring disease, the primary in which no cause is apparent.

Secondary hydrocele may be seen with any inflammation of the testis or epididymis, acute or chronic, and with testicular neoplasms. It is always present in acute infections. In chronic infections some fluid can nearly always be demonstrated in the tunica vaginalis, but whereas an amount sufficient to merit the name of hydrocele is seen in nearly all syphilic lesions, it is found in only 30 per cent. of tuberculous ones. An obvious hydrocele is uncommon in carcinoma.

The secondary hydrocele can usually be recognized as such on clinical grounds alone. It is small in size, slack, and can be transilluminated easily. It is of no importance in itself, but is important in that it conceals the outline of the testis. If, therefore, the nature of the lesion is in doubt, the hydrocele should be tapped to allow the testis and epididymis to be examined.

A primary hydrocele is the commonest of all scrotal swellings. Sometimes the trouble can be traced to a blow on the testicle, but usually the cause is quite unknown. It is tempting to suggest that the repeated trauma of clothing may be responsible, but the naked savage is just as liable to the complaint as his trousered brother and far more proud of it. The first onset is commonly in later middle life, from 40 to 60. Primary hydroceles are usually large, tense and fairly thick-walled when first encountered, because they have been present some time. They are symptomless, and the patient comes for advice only because the weight of the swelling annoys him, or its size attracts attention.

A hydrocele is seldom a regular ovoid, but usually shows a circumferential constriction. If the swellings each side of the waist are equal the shape is that of a monkey nut, if the upper one is smaller that of a pear. The testis, which cannot be felt but whose shadow can be demonstrated with a torch, should lie below and behind, but in about 1 per cent. of men it lies in front, and very occasionally above. A primary hydrocele is tense, so that it may be said to feel elastic rather than to fluctuate. It transilluminates, but not well, for its coverings are thickened, and a good light is needed.

The fluid, when withdrawn with a trocar, is clear, deep golden yellow, and heavily charged with albumin, so that it feels soapy on the fingers, froths as it pours into the receiver and goes solid, almost like white of

egg, when boiled. It has a specific gravity of 1020 to 1025. The fluid contains fibrinogen but no thrombin, so that it clots when blood or tissue fragments are added, but not before. In old-standing hydroceles the fluid is often dark brown and contains cholesterin crystals in suspension, giving the appearance of gold paint.

A swelling of the tunica vaginalis containing blood is a hæmatocele. The most important hæmatoceles are those which contain blood-stained fluid rather than blood, for they are secondary to a lesion of the testicle, a new growth or a torsion. In such cases the swelling may transilluminate, and it is not until fluid has been withdrawn that the presence of blood is recognized. Hæmatoceles containing pure blood are nearly always the result of trauma, though they may follow the spontaneous rupture of a vessel in the cord. If the blood is fresh they give the shape and sense of fluctuation of a hydrocele, but they do not transilluminate ; the diagnosis is made on the history and those physical signs, and is confirmed by tapping. If the blood is old and clotted, the hæmatocele presents as a solid testicular tumour without anatomical features ; it cannot be differentiated with certainty from a malignant tumour, and must be treated by orchidectomy.

Cysts of the epididymis must be considered in conjunction with hydrocele for two reasons.

First, because these two conditions, which have no characters in common, except that both are scrotal swellings that transilluminate, are constantly being confused. The smart candidate who comes to Queens Square talking of streptomycin, protein hydrolysates, and the tetralogy of Fallot, has only to be led to a spermatocele to suffer instant deflation.

Second, because epididymal cysts have no clinical resemblance or pathological relationship to the other lesions of the epididymis, which are inflammatory.

The main features of the two swellings may be contrasted :—

	HYDROCELE	CYST OF EPIDIDYMIS
SHAPE	Smooth, ovoid	Group of irregular knobs.
TENSION	Tense	Usually soft.
TESTIS	Not felt	Can be felt separate from the swelling. An epididymal cyst of moderate size may feel like another testicle.
TRANSILLUMINATION	With some difficulty at any rate in the older hydroceles.	Shines like a Chinese lantern.
FLUID	Clear yellow High specific gravity round 1025 Goes solid on boiling Microscopy : no cells, often cholesterin crystals	Opaque and milky. Low specific gravity seldom over 1005. Slight cloud on boiling. Microscopy : spermatozoa usually mobile, occasionally dead, in more than 90 per cent.

References to a diagram of the structures which go to make up the testis and epididymis shows that, with the exception of the paradidymis or organ of Giraldes and the appendix epididymis, both of which are mesonephric remnants, the tubular structures from which cysts may arise communicate with the seminiferous system (Fig. 3). The great majority of cysts of the epididymis, therefore, contain milky fluid in which spermatozoa or their parent cells can be demonstrated. These spermatoceles are single, or contain groups of two or three cysts, and are usually large, at any rate larger than the testicle, when they are presented for diagnosis. They are unilateral and are seldom seen before middle life.

DUCTUS DEFERENS

PARADIDYMIS OR ORGAN OF GIRALDES (MESONEPHRIC)

APPENDIX EPIDIDYMIS (MESONEPHRIC)

VASA EFFERENTIA (MESONEPHRIC)

APPENDIX TESTIS OR HYDATID OF MORGAGNI (MULLERIAN)

SUPERIOR VAS ABERRANS OR KOBELTS TUBULES (MESONEPHRIC)

INFERIOR VAS ABERRANS OR VAS DEFERENS OF HALLER (MESONEPHRIC)

COMMUNICATE WITH SEMINIFEROUS TUBULES AND CONTAIN OPALESCENT FLUID AND SPERMATOZOA

APPENDIX TESTIS (HYDATID OF MORGAGNI)
SUPERIOR VAS ABERRANS
INFERIOR VAS ABERRANS
VASA EFFERENTIA

NO COMMUNICATION WITH SEMINIFEROUS TUBULES. CONTAIN CLEAR YELLOW FLUID

PARADIDYMIS OR ORGAN OF GIRALDES
APPENDIX EPIDIDYMIS

VESTIGIAL STRUCTURES ASSOCIATED WITH THE TESTICLE

Fig. 3.

Epididymal cysts containing clear fluid probably arise in the organ of Giraldes, since they are found in the head of the epididymis. They are

bilateral, multiple, each cyst seldom exceeding the size of a pea. They occur in older men and are usually found accidentally.

When palpation has demonstrated that the swelling is one of the testicle (using the word in its general sense), the condition of the epididymis and of the body of the testis must be examined separately, for the diseases of the two are distinct. The epididymis is the seat of acute and subacute infections, the testis of virus, spirochætal and malignant lesions ; and though disease arising in one may eventually involve the other, the pantechnicon diagnosis of " epididymo-orchitis " is no more accurate or useful in the surgery of the scrotum than is " Appendico-typhlitis " in that of the abdomen.

The swellings that involve the body of the testis are :—

1. *The orchitis of mumps.*—The testis, when affected, is usually involved about a week after the parotid. It is slightly enlarged, enveloped in a secondary hydrocele of moderate size, and tender, though not always very painful. The swelling is often bilateral.

2. *Syphilitic orchitis.*—Enlargements of the testis due to syphilis occur late in the disease, and are characterised by painlessness, loss of testicular sensation, and by the presence of a small hydrocele.

In congenital syphilis, orchitis is encountered in the second or third year. Syphilitic orchitis is the only common enlargement of the testis in infancy. The enlargement is hard and painless, and is described as producing a swelling the size and shape of a pigeon's egg. The syphilitic orchitis of infants seldom goes on to gumma, the final result being a small, stony-hard, and functionless organ.

Acquired syphilis may involve the testis at two periods. In the second, third or fourth year after infection a diffuse tertiary orchitis may be encountered, which resembles in every way the condition seen in the congenital form of the disease and, like it, ends in atrophy. Gumma of the testis is seen much later, usually about the tenth year, but sometimes as late as the 30th, and it is a localised rather than a diffuse process. The testis enlarges painlessly and later may present a localised knob, which softens and finally sloughs through the scrotal skin. At this stage the lesion may present as a typical gummatous ulcer, the scrotal skin irregularly punched out, the testis appearing in the bottom as a wash-leather slough ; or the sloughing testis may herniate through the hole in the scrotum and lie exposed on the surface.

3. *Tumours of the testicle.*—These are, almost without exception, malignant and they arise with equal preponderance in the body. The relation of non-descent to malignancy has been questioned by many writers but Campbell's figures seem to prove beyond any doubt that cancer is 43 times more common in the undescended or ectopic testicle than in the normal one. Campbell showed that in 9,741,097 Army

recruits the incidence of non-descent of the testicle was 0·23 per cent. If malignant disease is unrelated to non-descent, the incidence of non-descent in any series of malignant testes should also be approximately 0·23 per cent. In an analysis of 1,422 cases from the literature, however, he found 165, or 11·6 per cent., were in abnormally situated testes. This proportion agrees closely with that found by other writers, Rea 10 per cent., Gordon-Taylor and Wyndham 11·8 per cent., Dean 13·5 per cent., Miyagi 12·4 per cent., Hinman 12·2 per cent., and Rubaschow 11 per cent.

Two types of malignant tumour occur in the testis, the teratoma, and the seminoma, but on clinical grounds they cannot be distinguished. They are equally common. Both are seen most often between the ages of 20 and 40. Both are highly malignant. Both spread early by the lymphatic stream, often before the change in the testis has been noticed by the patient, above all to the paraaortic glands at the level of the first and second lumbar vertebræ, but also with the vas to the internal iliac glands. Both may show metastases in any part of the lymph system, locally in the lymphatics of the cord, or distantly in the supraclavicular glands. They are also carried by the blood stream to the lungs, bones, brain and liver. Both may give the Ascheim-Zondek reaction in the urine.

A malignant neoplasm of the testicle presents as an enlargement of the body of the testis, that is at first painless and may therefore remain unrecognized. The growth remains for some time within the confines of the tunica albuginea, so that it keeps its ovoid shape. The epididymis is progressively stretched over the tumour, first becoming a flat strap, later unrecognizable as a separate structure. There is usually a small hydrocele, occasionally a hæmatocele. The distinction from tertiary syphilitic or gummatous enlargement may be impossible on clinical grounds.

The seminomas are slightly commoner than the teratomas. They occur on the average in older patients, the mean age being 40, and with teratoma 28. While both are highly malignant, the seminomas are less so than the teratomas. There are many recorded instances of permanent cure following orchidectomy for seminoma, but few survivals after operation for teratoma. The reason for this difference is to be found in the proneness of the teratomas to disseminate very early by both the lymphatic and the blood streams, whereas the seminomas spread preponderatingly by the lymph channels, and the spread is longer delayed. Both seminomas and teratomas produce gonadotrophic substances which can be identified in the urine; they are most abundant, however, with the highly malignant teratomas, especially those of the " Chorion-epithelioma " type, with which they may attain a concentration equal to that of pregnancy. Both tumours are radiosensitive, but particularly the seminoma which is more so than any other malignant tissue.

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The differences between the two types of tumour are apparent only after removal. The seminoma on section is seen to consist of uniform creamy white tissue, interspersed with small areas of hæmorrhage and degeneration, that has replaced the normal testicular substance. Microscopically the tissue consists of large rounded cells, resembling the spermatogonia of the normal seminiferous tubules interspersed with occasional agglomerations of lymphoid tissue.

The teratomas, when cut across, present a very variegated appearance, and often seem to be demarcated sharply from the remaining testicular tissue, which may be displaced to one side or stretched over the tumour. The surface shows areas of hæmorrhage, many irregular cysts (hence the old term fibrocystic disease of the testicle) and islets of cartilage. The histological appearance of a teratoma is one of extreme complexity. Mature and embryonic tissues derived from all the germinal layers may be found, and there is a tendency for those which are associated in forming adult structures to appear together; thus squamous epithelium and fat, intestinal mucosa and plain muscle, or ciliated epithelium and cartilage, may be associated in the tumour. The metastases may contain all or most of the structures of the primary tumour, or one only.

Innocent teratomas, like ovarian dermoids may very occasionally be seen in children as irregular stationary, symptomless testicular lumps. They are common in horses. Sarcomas may occur in the testicle as in any part of the body, but they are very rare and the tumours formerly reported as sarcomas of the testis were seminomas.

The swellings of the epididymis are practically all inflammatory, and we must recognise four types of epididymitis, the gonococcal, the septic, the tuberculous and the non-specific. Gonococcal and septic epididymitis are acute infective lesions, with a short history and all the general and local signs of an inflammatory process. The whole epididymis is enlarged, firm and very tender. There is a small hydrocele, but through it the body of the testis can be felt to be normal. The scrotum on the affected side is red, hot, œdematous and tender. The two conditions cannot be distinguished on physical signs, and only the presence of a history of venereal infection, and of a urethral discharge in which gonococci are found, will give the clue. Gonococcal epididymitis usually occurs in the second or third week of the disease. Septic epididymitis follows prostatectomy, the passage of catheters or operations on the urethra. Gonococcal epididymitis usually subsides without suppuration, that due to pyogenic cocci is more liable to end in abscess formation.

Tuberculous epididymitis differs from the above in that it is subacute, and that it usually affects part of the epididymis only when the case is first seen, and does not involve the scrotum. In a typical case a firm and very tender nodule is discovered, usually in the globus minor, but not uncommonly in the globus major. An effusion into the tunica vaginalis

is unusual. Later the process spreads throughout the epididymis, the nodules coalesce and break down, the scrotal skin becomes adherent, and finally a cold abscess discharges at the back of the scrotum, leaving a persistent sinus.

Tubercle bacilli can undoubtedly reach the epididymis by the blood stream, along the lumen of the vas, or by the perivasal lymphatics. Which is the common route is still undecided. General surgeons on the whole, including men who have studied large series like Hamilton Bailey and Illingworth and Dick, favour embolic infection by the blood stream. Urologists believe that in the great majority the epididymis is involved secondarily from a focus in the prostate, and they point to the common observation that the whole genital duct system on one side, epididymis vas and seminal vesicle, is usually demonstrably diseased when the case is first seen, and to the frequency with which the second epididymis is involved in sequence, in support of this view.

The diagnosis of tuberculous disease of the epididymis on the basis of the physical signs of a scrotal swelling can be no more than presumptive. Confirmation must be sought by examination of the urine for tubercle bacilli, by an investigation of the whole genito-urinary tract, and by scrutiny of other sites, particularly the lungs, in which a primary tuberculous focus may lie. Until proof is obtained a nodule in the epididymis is shelved but not labelled. It may be tuberculous, it may be due to an unidentified infection, it may even be malignant.

Non-specific epididymitis is a term that includes all those swellings of the epididymis that cannot be identified as gonococcal, septic or tuberculous. Some are due to mumps, some to low grade gonococcal infections which are difficult to identify, some follow a mild urethritis of a few weeks duration which is probably venereal, but is entirely unresponsive to sulphonamides or penicillin and has by some been attributed to a virus infection. My friend V. E. Lloyd labels this group " Epididymitis Obscura," a diagnosis that implies ignorance and interest but no more, but Sampson Handley would retain " Non specific Epididymitis " because it does at any rate imply an assumption that the lesion is an infective one.

Non-specific epididymitis is a common complaint in active young men. Handley quotes the Army Medical Dept. Bulletin of 1943 as reporting that in a six-month period there were 680 cases of Epididymitis in Army records of which 79 were gonococcal, 19 tuberculous, and 582 non-specific. Non-specific epididymitis resembles the tuberculous lesion, which should always be assumed till it has been disproved, in that it presents as a hard, tender enlargement without the signs of an acute inflammatory process and without involvement of the scrotum ; it differs in being smooth rather than nodular, and in subsiding with rest alone.

The one testicular event which involves both the body and the epididymis is torsion. Torsion never occurs in a testicle that is normally

descended and fixed. The incidence in undescended and in scrotal testicles is about equal, but since only one testicle in 400 remains undescended the incidence in the undescended is four hundred times as great as in the descended.
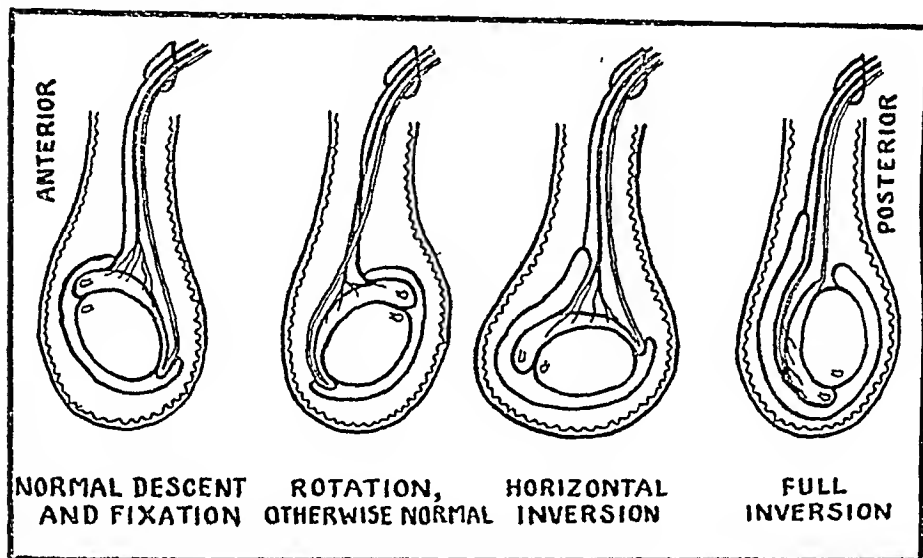
Fig. 4.

The normally descended and fixed testicle hangs in the scrotum with the epididymis behind and the body in front, the upper pole tilted about 30 degrees forward (Fig. 4). The body except for the mediastinum and a narrow strip in contact with the body and tail of the epididymis, and the globus major and sides of the epididymis, are clothed by the visceral layer of the tunica vaginalis, but the back of the cord and of the body and tail of the epididymis have no serous covering. Such a testis has no free pedicle and cannot therefore undergo torsion. Three abnormalities are encountered, rotation, horizontal inversion and full inversion. The rotated testicle has the epididymis in front but is normally clothed by the tunica vaginalis; it cannot undergo torsion, but it may suffer injury in the tapping of a hydrocele unless the rotation has been discovered. The horizontally inverted testicle lies with the body horizontal and the epididymis above; the cord enters the body of the epididymis and the tunica vaginalis covers the back of that structure in its upper half. The fully inverted testis is upside down and the cord enters its lower end, so that the whole epididymis except the tail is covered with serous membrane. Thus the horizontally and fully inverted testicle hang in the tunica vaginalis by a narrow vascular pedicle, which may become twisted. The undescended testicle usually lies in a patent processus funicularis, but the back of the cord and epididymis may be entirely extraperitoneal as they are in the normal testicle when the processus is closed. In some however a testis and epididymis normally arranged are suspended free by the

spermatic cord, so that torsion may occur at the cord (Fig. 5). In a few the normal folding of epididymis on testis has not occurred, and the testis is suspended by an elongated epididymis, so that torsion may occur either at the point when the vasa efferentia join the epididymis, or in the body of the epididymis.
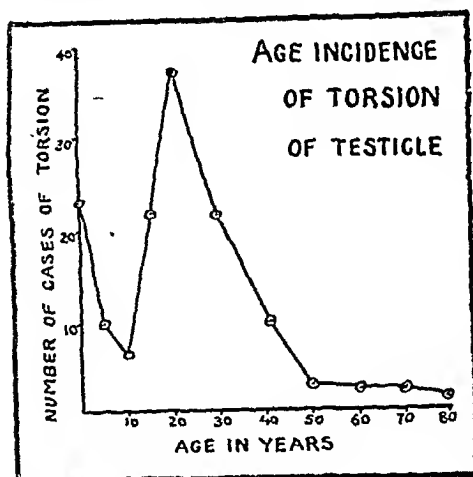
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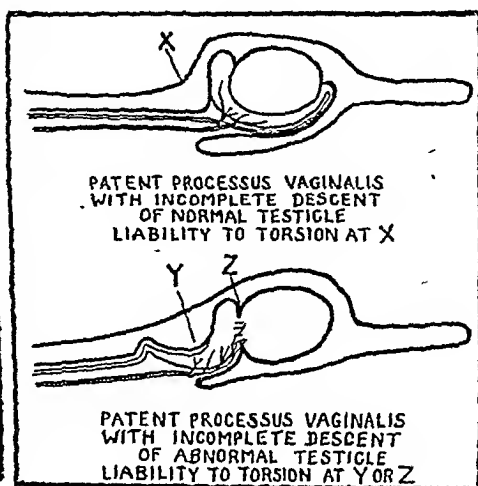
Fig. 6.

Torsion may occur in a malfixed testicle at any age, but it is most commonly encountered at two periods between 5 and 10 and between 15 and 20 ; it is rare after 50 (Fig. 6). The exciting cause is probably a sudden impact of the thigh on the testis, because the direction of torsion is nearly always from without inwards. The onset of the torsion is usually dramatically sudden, accompanied by pain, vomiting and shock. If the patient is examined immediately after the incident, a tense and very tender swelling, which lacks the outline of the normal testicle and is usually higher, will be found; later the pain and tenderness are less, but the swelling is further obscured by the development of a hæmatocele and the scrotum over it shows a cold œdema.

The surgeon is usually called when the torsion is fixed and the testicle already dead. Brigadier Fettes, Consulting Surgeon to the Army, has recounted an occasion when he was more fortunate.

" A civilian engineer on tour to Calcutta, came to spend the night at the Wheeler Club. He went to bed in the next quarter to mine at about eleven. Two hours later I was awakened by someone knocking at my front door. I found the engineer on hands and knees, ashen pale, dripping sweat, in unbearable pain, which had come on when in bed, and had lasted about half an hour before he came for help. I went on my knees and stood him up in front of me, risking his collapsing, but in that position, if the untwisting is successful, the mechanism of the process is well demonstrated. It is an even chance that one tries to untwist the

wrong way, the slightest rotation then meets resistance, and achieves nothing except to increase the pain, whereas reversing the process is easily possible. In such circumstances one does not count the number of turns one untwists, but in the process the testis descends for an inch or more, till it reaches its normal level, and the pain is gone.

I once had a watch I used to fiddle with, hanging it like a pendulum on its chain, twisting it round till the links tightened, the chain coiled on itself, shortening in the process, and up rose the watch. In reverse, the coils came undone, the chain lengthened, and down came the watch.

The engineer continued his journey next day, determined at the first possible opportunity, to submit to any operation which would prevent a recurrence."

The treatment of scrotal swellings is beyond the scope of this lecture, but I would remind you that when that treatment is operative, the right approach to scrotal contents is through the scrotum. The approach is a direct one; the incision through the raphe provides one of the few instances in the body where no visible trace remains of the surgeon's handi-work; the scrotal skin stretches for access and contracts for suture; drainage, if required, is dependent; and healing is rapid and uneventful. The median septum provides the perfect physiological grasp for retaining the surgically descended testis—non traumatic, yielding, yet always resisting and finally overcoming.

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THE OCULAR SEQUELÆ OF HEAD INJURY

Lecture delivered at The Royal College of Surgeons of England

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by

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THE MOST IMPORTANT consequence of an injury of the head is the damage which results to the brain, but lesions occur in many other structures and a description of the ocular sequelæ demands that consideration be given to fractures of the skull and to ruptures of the meninges and vascular channels, while injury to the orbit and its contents must also be included. The ocular sequelæ of head injury may be classified as : (1) Signs which are important as a guide to general treatment ; (2) Other immediate sequelæ ; (3) Remote sequelæ ; (4) Blindness.

1. Signs which are Important as a Guide to General Treatment

Cases of head injury may recover spontaneously or they may require surgical treatment. The surgeon must decide whether to operate, and, if so, when this should be done ; furthermore, he has to make a decision regarding the localisation of his surgical sortie. Assistance in the solving of these problems may be derived from a study of the eyes.

(a) *The Pupils.* Variation of the pupils of the eyes in shape and size is common after head injury. This may be the result of direct trauma applied to the globe, the so-called traumatic mydriasis, in which small ruptures of the pupillary margin of the iris are often found or it may be the result of a lesion of the optic nerve or oculomotor nerve. In some cases, however, the pupils are seen to be normal immediately after the injury, and, after preliminary contraction, one pupil is subsequently observed to dilate. This may be followed by dilatation of the pupil of the opposite side. This sign is an indication of extradural or subdural hæmorrhage on the same side as that of the first dilated pupil. It is thought to be due either to pressure on the midbrain or to cortical stimulation. Hemiplegia may be found on the side of the first pupil to dilate and is due to displacement of the midbrain by the hæmorrhage and to resulting contusion of the opposite cerebral peduncle against the sharp edge of the tentorium.

(b) *Deviation of the Head and Eyes.* Deviation of the head and eyes towards the injured side is a useful localising sign of a destructive lesion of the base of the superior and middle frontal convolutions, provided the same convolutions are intact on the opposite side.

(c) *Papillœdema.* This is an uncommon sign in head injury, and was found by Cairns in only 9 per cent. of his series. Its appearance is usually delayed until about the fifth day after injury and it suggests that craniotomy may be required, even if the general condition of the patient is, at that time, fairly satisfactory. It is not, as a rule, well marked, being limited to slight blurring of the disc margins with slight congestion of the central retinal vein. The cause is either blood clot, which may be extradural or subdural, cerebral œdema, collections of cerebral spinal fluid mixed with a varying amount of blood in the subdural space, or cerebral abscess after compound fracture. Marked papillœdema may occur as a result of hæmorrhage into the sheath of the optic nerve, and in such a case the vision may be seriously embarrassed. The mechanism of formation of the fundus appearance in this condition is similar to that of papillœdema due to raised intracranial pressure, being due to pressure on the vessels in the subarachnoid space causing obstruction of the central vein of the retina, but not of the central artery of the retina. It is considered that, in cases of subarachnoid hæmorrhage, papillœdema may occur in a similar manner, and that the intraocular hæmorrhages, particularly of subhyaloid type, which are characteristic of this condition, are due to rupture of branches of the central vein of the retina due to increased venous pressure. Harrison Butler has, however, reported a case of subarachnoid hæmorrhage in which he saw blood appearing at the edge of the optic disc as though percolating through the margins of the cribiform plate from the subarachnoid space.

2. Other Immediate Sequelæ

(a) *Visual Tract.* This is frequently injured in some portion of its course, but the optic nerve and the optic radiations in the occipital lobe are damaged more often than the other parts. Injury of the optic nerve is observable clinically by atrophy at the disc and this may be visible within 12 days. The degree of pallor does not bear any direct relationship to the visual acuity which may vary from inability to perceive light to nearly 6/6, with a similar pale appearance of the disc. When any degree of vision remains some loss of field can always be demonstrated. Hughes has analysed field defect after indirect injury of the optic nerves into two groups. The first or typical group shows some loss of the inferior field with general depression, and the upper nasal field is the least affected. The second group shows irregular peripheral or central defects which cannot be co-ordinated. Arcuate scotomata may be found in either group. The cause of optic nerve lesions after head injury has always been controversial. It has been considered in the past that they are due to mechanical damage to the nerve by bony fragments resulting from fracture of the optic canal. There seems no doubt, however, that only a small proportion of such cases, probably about 10 per cent., have a fracture of the optic canal. Contusions or stretching of the optic nerve have been said to be responsible, and it may be that they play a part,

but it appears that the immediate exciting cause is spasm or damage of the nutrient vessels. The presence of arcuate scotomata favours such a theory, and it is recognised that obstruction of the central artery of the retina can be induced by trauma. Some recovery of vision or of field may occur up to four months after injury of the optic nerves, but it is most marked in the first four weeks. Alternatively, progressive deterioration may be found to proceed for several months. The optic chiasma can be involved in injuries of the front of the head, and this is often associated with fracture of the posterior wall of the frontal sinus and of the roof of the orbit. The characteristic lesion is bitemporal hemianopia with sparing of the maculæ and it is interesting to note that residual islands of vision may occur in the otherwise lost temporal fields as may be found in cases of pituitary tumour and in chronic glaucoma. The lesion also appears to be the result of damage of the nutrient vessels rather than trauma from fracture fragments. The optic tracts may be affected in head injury but this is not common. Congruous field defects where no focal lesion of the occipital lobes has been demonstrable by the electro-encephalogram, have been thought to be due to tract lesions. The occipital lobes are frequently damaged in injuries of the head, but in the closed type this tends to be localised to the outer parts, as the calcarine portion is protected. Visual defect is therefore small unless infection occurs. Perforating injuries may cause any type of lesion and during the two world wars there have been unique opportunities to correlate visual field defects with the site of damage. Possible bilateral cortical representation of the macula has been a subject of repeated controversy, but accurate observation during the recent war has provided no proof that this occurs. Observations by Hine, in the first world war, on patients with mild degrees of closed head injury showed complete homonymous hemianopia immediately after the receipt of the trauma. This was shown to be transient and to clear completely. It may be that many cases of injury to the head would show this condition if perimetry were possible at an early stage. Such defects of the higher visual functions as alexia, visual object agnosia, loss of topographical memory, and loss of visual imagery may occur after head injury if the lesion is on the left side of the cerebrum in right-handed persons, or on the right side in those who are left-handed.

(b) *Oculomotor Palsies.* Injuries of the extraocular muscles or their nerve supplies with resulting diplopia are frequently found in association with head injury. It must be emphasized that in the majority of cases there is not a complete paralysis; defective action of a muscle rather than complete loss of function is the usual finding. The lesion occurs at the time of the injury, but the diplopia may not be noted for some considerable time if one or both eyes are bandaged, if a hæmatoma of the lids with resulting closure is present, or if a ptosis has been induced by paresis of the levator palpebræ muscle. The external rectus muscle has, in the past, been considered to be that which is most frequently affected, due to damage to the abducens nerve against the sharp upper border

of the petrous portion of the temporal bone, but figures obtained from a series of cases at the Military Head Injuries Hospital during the recent war have revealed that both the superior rectus muscle (24 per cent.) and the superior oblique muscle (21·5 per cent.) are more commonly affected than the external rectus muscle (11 per cent.) (Table I).

TABLE I

ANALYSIS OF OCULOMOTOR MUSCLES AFFECTED AFTER HEAD INJURY.

Paresed muscle, muscles, or nerve trunks	Closed injury	Open injury	Total	Percentage
Superior rectus	33	0	33	24
Superior oblique	30	0	30	21·5
External rectus..	15	1	16	11 ·
Inferior oblique	8	0	8	6
Internal rectus ..	6	0	6	5
Inferior rectus ..	2	1	3	2
Total III and IV cranial nerves	2	0	2	1
Total III cranial nerve	6	0	6	5
Inferior division of III cranial nerve ..	3	0	3	2
Various combinations of muscles without nervous relationship ..	30	1	31	22·5
Totals ..	135	3	138	100

A further analysis of cases has shown that when there is no evidence of orbital injury the trunks of III, IV and VI are predominantly affected, whereas when the orbit is injured single muscles supplied by III or combinations of muscles without nervous relationship are defective (Table II).

TABLE II

EVIDENCE OF LOCAL ORBITAL INJURY

	Evidence of orbital injury	No evidence of orbital injury
Superior rectus ..	21	12
Superior oblique	7	23
External rectus ..	2	14
Inferior oblique	8	0
Internal rectus ..	2	4
Inferior rectus ..	2	1
Total III and IV cranial nerves	2	0
Total III cranial nerve	2	4
Inferior division of III cranial nerve	3	0
Various combinations of muscles without nervous relationship ..	22	9
Totals ..	71	67

This indicates that orbital injury is frequently responsible for oculomotor paresis, and the very rapid recovery of muscle balance which may occur in patients who have such pareses after head injury indicates that bruising of the muscle or a nerve lesion close to the muscle has been responsible. Chavasse has pointed out that, following the paresis of an extraocular muscle, secondary changes occur in other muscles. These are overaction of the contralateral synergist, contracture of the direct antagonist, and inhibitional palsy of the contralateral antagonist. These changes should be remembered because sometimes they tend to cause continuation of symptoms in spite of spontaneous recovery of the muscle originally damaged. These changes may occur rapidly and may be clinically obvious within 14 days after injury. The normal course of oculomotor paresis after head injury is one of spontaneous recovery, though this may take many months to complete. Sometimes function returns early and completely, while in others there is a long interval before recovery commences. Some patients appear to regain the power of ocular movements, but to retain a " habit " of diplopia which persists in the absence of treatment. A minority of cases do not show organic functional recovery. Persistent diplopia is finally relieved by neglect of one image and the final condition approaches uniocularity and comitance. It is most important to realise that there is a high incidence of psychoneurosis in cases of diplopia, and the successful management of these patients demands that this aspect is borne in mind throughout. Orthoptic methods are the principal form of treatment, and they can be commenced as soon as the person becomes ambulatory. Such treatment hastens recovery in patients who would otherwise recover spontaneously, and it causes function to be fully regained in some people in whom it would not otherwise occur. It shows its most striking successes in the group of patients who have recovered ocular movement without losing their diplopia, when one or two sessions of treatment may be sufficient to restore normal binocular vision. It should be pointed out that in treating these patients the Orthoptist avoids exercising the weakened muscle. The binocular vision is stimulated in other parts of the field of movement so that, as recovery of the paresis occurs the individual is enabled to employ it to full advantage. Cases which do not recover spontaneously in 9-12 months should be treated by surgical means, according to the accepted principles for the treatment of paretic strabmismus, or, if this is contra-indicated, the incorporation of suitable prisms in spectacles may be useful. The use of a frosted glass in front of one eye may be necessary in some cases of great severity. The Hess screen is most valuable in the management of these cases for recording the condition of the oculomotor muscles at the different stages of recovery.

(c) *Ptosis.* This may occur after head injury as a result of a complete paresis of the oculomotor nerve, and in such a case it may serve to prevent diplopia. It may be the result of direct trauma affecting the levator palpebræ muscle, or, in mild degree, to damage of the sympathetic nervous

system. It may occur in transient form due to hæmatoma. Many of these cases recover spontaneously, but where the condition persists surgical treatment may be required. Blaskovic's operation is contra-indicated if the levator palpebræ muscle is paralysed and elevation by a fascial graft may be necessary. It is, however, not easy to regain a completely natural appearance by this latter method.

(d) *Defects of Convergence.* This is one of the most common ocular sequelæ of head injury, and it may occur with or without defect of accommodation. It may be the result of damage to the oculomotor nerve, while in cases of paresis of one of the vertically acting extraocular muscles it can easily be understood to be due to " absence of reward." Some patients may have had this defect before their injury, and in others it is associated with severe psychoneurosis, but in the majority of cases it appears to be induced in uncomplicated form by the injury, and its exact cause is a matter of some doubt. It is suggested that it is a manifestation of the extreme fatigueability which affects the whole body after severe injuries to the head. Convergence may not be possible in the early stages after recovery of consciousness, due to the fatigued state of the internal rectus muscles, and a habit is formed, which may be exaggerated by the suggestion that the eyes have been damaged, and which becomes perpetuated. It has been observed that some hypermetropes, after injuries to their heads, can converge when wearing their glasses, but without glasses the combined effort of accommodation and convergence is more than they can undertake. Some patients recover spontaneously from this defect, but, in them, the process is hastened, and in others it is brought about by orthoptic treatment, and the rapidity of cure in most cases is an indication of the functional nature of the defect and of the absence of organic cause. This treatment should not, of course, be commenced until the stage of fatigue is over and the patient can leave his bed.

(e) *Pupillary Defects.* An Argyle Robertson pupil may be seen in injuries which involve the base of the brain, and a similar type of pupil may occur in lesions of the efferent pathway of pupillary contraction. A second efferent path is postulated, in addition to that through the ciliary ganglion, which is associated only with contraction of the pupil to accommodation. It passes by way of the inferior branch of the third nerve, the nerve to the inferior oblique muscle, and the episcleral ciliary ganglion, and may continue to function after the primary route is severed, and cause a pupil which contracts to accommodation and not to light. Irregularity of the shape of the pupils after cerebral contusion is a recognised abnormality but the mechanism is uncertain. It may be the result of irregular contraction of the constrictor pupilli muscle due to traumatic changes of the third nerve or its nucleus.

(f) *Nystagmus.* This may occur as a transient or a permanent phenomenon. The former is usually the result of damage to the brain stem, and the latter occurs with a lesion of the vestibular apparatus.

(g) *Surgical Emphysema of the Orbit.* This may occur in a head injury which involves fracture of the orbital aspect of the nasal sinuses. Proptosis may result, but is usually transient, and causes no serious sequelæ.

(h) *Dislocation of the Globe.* The globe may be dislocated on to the face, into the antrum of Highmore, or into the cranial cavity in severe head injuries involving the face.

3. Remote Sequelæ

(a) *Arteriovenous Aneurysm.* This is usually the result of an injury of the internal carotid artery where it passes forwards in the outer wall of the cavernous sinus, and it usually develops weeks or months after the original injury. The lids swell, the conjunctiva becomes chemotic, and the conjunctival veins are dilated. Some degree of proptosis occurs, and sometimes this may be so gross as to cause keratitis from exposure. Pulsation of the globe may be visible, and a bruit may be heard with the stethoscope. The patient may be conscious of the sound of this bruit and it may cause great distress. Oculomotor paresis is usually present, the pupil may be dilated, papillœdema may be seen, and, if the condition is allowed to persist, optic atrophy may ensue. Intense pain sometimes occurs. Treatment may require ligature of the common carotid artery after prolonged initial compression and under local anæsthesia. It is advised that this should be done in such a manner that the ligature can be removed if the patient shows signs of becoming comatose. It should be noted that pulsation of a globe may occur by transmission from the brain through a fracture of the orbital roof.

(b) *Delayed Oculomotor Paresis.* This may be the result of inflammatory changes resulting from infection ; it may follow callus formation in the healing of fractures, and it frequently occurs with arteriovenous aneurysm.

(c) *Arachnoiditis.* A history of head injury has been found in about 10 per cent. of cases of this condition of thickening of the arachnoid and pia and it is considered that trauma may be a predisposing, though not an exciting cause. Compression or spasm of the blood vessels supplying the optic nerves and optic chiasma occurs with resulting visual defect, and there is demyelination and atrophy of optic nerve fibres which may proceed to blindness. Loss of vision may be sudden or gradual and progress tends to be capricious. X-ray and cerebrospinal fluid examinations reveal negative findings and visual field defects are very variable, though a very irregular peripheral limit is said to be characteristic, and central scotomata are commonly found.

4. Blindness after Head Injury

This may occur as a result of injury to the occipital cortex and in some cases when the tips of the occipital lobes are involved a central scotoma is found, with some preservation of the peripheral fields. Some cases

have been seen during the recent war in which only the central field to about 5 degrees from fixation remains in each eye. These cases give rise to the suggestion that a hysterical factor may be present. Many skull wounds give rise to extreme optic atrophy, some of which are post-papillitic in type, while in a number of cases of bilateral optic atrophy no cause has been found other than a history of relatively mild head injury. It is possible that some of these may be cases of arachnoiditis. Injuries of the anterior region of the skull may cause blindness and of these the commonest are the " through and through " injuries which sever both optic nerves, or damage the region of the chiasma, but smash wounds of the fronto-orbital region may also result in loss of vision.

The ocular sequelæ form a large and important part of the results of head injury and co-operation between neuro-surgeon and ophthalmologist throughout the period of recovery is desirable to ensure the most satisfactory results.

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THE GENESIS AND SPREAD OF
MAMMARY CANCER

Lecture delivered at The Royal College of Surgeons of England

on

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by

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IT IS SOMETIMES said that pathology has little further contribution to offer to the problem of mammary cancer, and it must be admitted that no tumour has been so intensively studied during the past hundred years and more. Ever since the publication of Sir Astley Cooper's and Brodie's work in this country, a constant stream of literature has covered all aspects of the subject, diagnosis, prognosis and therapy, as well as much experimental work. Yet the general attitude to cancer of the breast remains pessimistic. Diagnosis at the early, curable stage is difficult; prognosis is uncertain and the present variety and combinations of methods of treatment suggest still a phase of therapeutic trial and error. Recorded, acceptable, all-over, five-year survival rates rarely exceed 35 per cent.

This unsatisfactory state of affairs is being vigorously attacked from various angles. As a pathologist, I think that much can still be learnt from further study of mammary tumour tissue and, by retracing the evolution and growth of malignancy, more knowledge may be gained about its nature and possible causation. As Billroth remarked, " die Mamma ist die Amme der Geschwulstlehre." Until the intrinsic cause of cancer in the human subject is discovered, effective attack on the disease in any organ is mainly limited to accurate diagnosis and appropriate treatment. When the patient with mammary cancer is first examined, the disease, in the majority of cases, is already well established, but this does not lessen the value of a recognition and understanding of the genesis and early stages of the process and justifies re-examination of the changes which lead to cancer and its spread in and beyond the mammary area.

GENESIS

Cancer may be described as the end-stage of a proliferative activity which produces malignant cells. Sir Jonathan Hutchinson, one of the great Presidents of this College, when lecturing in the Royal Infirmary, Edinburgh in 1908, spoke of " lunatic colonies of cells in which, without

rhyme or reason, proliferation has become the order of the day." He was not discussing cancer cells, for he added, " I invite you to think more kindly of these unfortunate cells, and to believe that they must have experienced, in the first instance, much provocation." A study of this " much provocation," leading to proliferation and malignancy, lies at the root of all cancer work. Proliferation, however, needs definition, for in mammary tissue there are two types of epithelial activity which are different in kind, in pattern and in outcome and they need two descriptive terms. For many years now, in this Laboratory, we have used the terms *adenosis* and *epitheliosis* and they need, I think, no apology, for much confusion in the interpretation of mammary pathology, especially in experimental work, has been due to not recognizing and distinguishing them.

Adenosis describes the glandular growth which produces more and larger lobules, that is, a proliferation by branching of the lobular tree (Fig. 1). It is essentially a physiological response to growth stimuli, though it may be exaggerated, irregular and even localised in a mammary area. It is seen most characteristically in pregnancy. *Epitheliosis* describes the multiplication of epithelial cells within existing gland structures, without the formation of new gland elements ; it is a cellular filling of the lobule (Fig. 2), not a branching, and indicates a more or less pathological response, though it may be only a temporary activity and be followed by cell degeneration and cyst formation.

All epithelial proliferation in mammary tissue is of one or other of these two types and the important question is their relation to tumour growth. Normal growth in the breast, as seen in the slow development of structure from birth to maturity, is essentially an adenosis, but until puberty, the glandular structure remains a rudimentary framework and any type of pathological activity is exceedingly rare. As far as I know, no carcinoma is recorded. From puberty to maturity new glandular tissue is produced by budding from the ducts already laid down and lobules are gradually built up. This glandular increase or adenosis is associated with considerable growth of stroma and some fat, until the adult organ is formed. During this period, roughly 15 to 25 years, adenosis may produce an adenoma, usually a fibroadenoma, localised or, very rarely, diffuse. The likelihood of either carcinoma or sarcoma at this period is negligible. I, myself, have seen only three cases of carcinoma under 25 years in many hundreds of breast cancers examined ; they were quite exceptional, but microscopically they showed no genetic relation to the lobular branching which had formed the mature breast. Early pregnancy shows great increase of glandular tissue, that is, a very marked adenosis, to provide the extensive secreting surface needed for lactation. When lactation ceases, the secreting structures degenerate and gradually disappear. Each subsequent pregnancy involves a new adenosis for a new lactation. Tumour formation is very rare during pregnancy or

lactation. Only 1·5 per cent. of human mammary cancer was associated with reproduction in a large cancer series I investigated in Edinburgh. But diagnosis at these periods is difficult, because tumour is masked by physiological mammary enlargement, and prognosis is highly unfavourable. I have found no evidence to suggest that cancer originates as an adenosis in the secreting breast; in all the cases examined it began in the ducts as an epitheliosis and showed destruction of secreting tissue, with rapid growth and spread and an early fatal outcome. A fibroadenoma present when pregnancy begins will increase in size and secrete during lactation, but this is very rare, as the tumour is usually removed earlier.

The climacteric and menopausal period is our chief concern. This is "the cancer age" of the breast, for all age-frequency figures show that malignant growth has its highest incidence in this period and especially between 50 and 60 years. There is much histological evidence to support the view that conditions which favour abnormal cell activity are particularly associated with mammary involution and atrophy. This involution is a slow process but so, also, is cancer formation. If we accept the time analogy with occupational cancer, where 5 to 15 years, or, on an average, 10 years are needed for malignant growth to become clinically evident, the 10 years *before* the highest incidence period for mammary cancer would be indicted as the "dangerous ages," that is, the climacteric or involution period of roughly 40 to 50 years. Ovarian function is then subsiding and with it, ovarian and pituitary control of normal mammary activity.

Examination of climacteric and menopausal mammary tissue suggests the possiblity, if not indeed the probability, that during involution every breast shows, in varying degree, some proliferative activity. Such activity may or may not be clinically evident, as enlargement and pain. One would not expect, however, to find adenosis, which produces new glandular tissue, to play any part in the climacteric and menopausal retrogressing breast, but in almost all cases this does seem to happen. It may be explained by an endocrine instability, with compensatory pituitary hyperfunction and stimulation. This finds some confirmation in the fact that menopausal patients may give a mildly positive Aschheim-Zondek reaction. Menopausal adenosis may be generalized, with or without clinical enlargement; if localised, it may present a doubtful clinical lesion of focal induration, even when, microscopically, the area shows only lobular overgrowth of physiological pattern, indistinguishable from an early pregnancy proliferation (Fig. 1). In some cases, minute, multiple fibroadenomas are also formed by an accompanying stroma activity in the lobules.

This climacteric adenosis used to be called chronic lobular or chronic glandular mastitis, or, by Ewing, chronic productive or chronic adenomatoid mastitis. Bloodgood called the condition senile parenchymatous hypertrophy, though Hertzler envisaged some embarrassment in telling

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a patient whom he described as "a husky dame of 45 or so" that she was suffering from anything senile! There is no inflammatory basis in the ordinary sense, and for an involuting, fibrosing tissue, the terms fibroadenosis or fibroadenomatosis, or if cysts are also present, cystic fibroadenosis, are more acceptable and self-explanatory. Later, as hormonal stability is re-established, this glandular proliferation subsides, the hypertrophic lobules gradually fade out with some hyaline change and, eventually, in senile tissue, only scattered small ducts and some cystic elements remain. Ewing noted that fibrosing adenomatosis, as he later called the condition, gives rise to a mistaken pathological diagnosis of cancer more often than any other lesion in the breast. Where no other type of activity is found, such cases do not show "recurrence" even after limited resection, as Ewing and Bloodgood testified. The essentially benign nature and later atrophic character of this proliferation may be accepted and we can dismiss adenosis, even at the menopause, as a type of activity which leads to mammary carcinoma. In other words, in the human breast at least, adenosis does not become adenocarcinoma. In rare cases adenosis may lead to sarcoma, through the intermediate phases of fibroadenoma and fibroma.

Climacteric adenosis is, however, not always a pure condition and, in fact, may be rarely so. If a clinical tumour is evident, adenosis is usually found in association with epitheliosis and cysts and it is these features which make enlargement of the breast suspect at this period. There is, mercifully, an evolutionary tendency to normality in the breast, as in all tissues and in the majority of cases, proliferation of every type subsides with fibrosis, fat deposit and small cyst formation, so that permament quiescence and atrophy supervene. But in a minority of cases proliferation is persistent and progressive, and cancer is produced. We must look for its genesis, therefore, in the second type of proliferation, epitheliosis (Fig. 2).

Epitheliosis occurs primarily in the ducts, at any level, though it may spread to the smaller branches of the lobule, if these are still intact. Later, the proliferated cells in the duct tend to degenerate, with the formation of cysts. The problem of cyst formation is still somewhat obscure. There is some justification for that very dangerous clinical diagnosis, a "milk cyst," for the breast as part of the reproductive system, is liable to what may be called physiological trauma, especially inadequate or want of lactation, as well as ill-defined cyclic changes, hormonal dyscrasias and inflammatory lesions. Normal structure may, therefore, already be impaired before mammary involution sets in with the climacteric and on this distorted tissue background would be imposed the further changes which may lead to cancerous growth. Cysts may also be formed by simple duct dilatation but the majority are apparently produced by cell proliferation within the duct, that is, by epitheliosis, followed by cell degeneration. Cysts differ in type, as cell degeneration may take different forms.
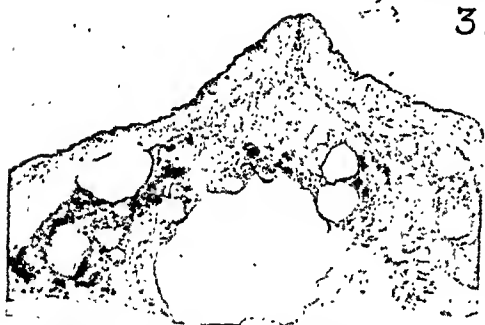
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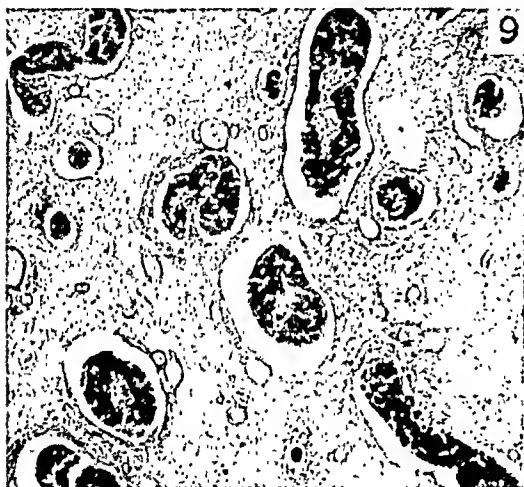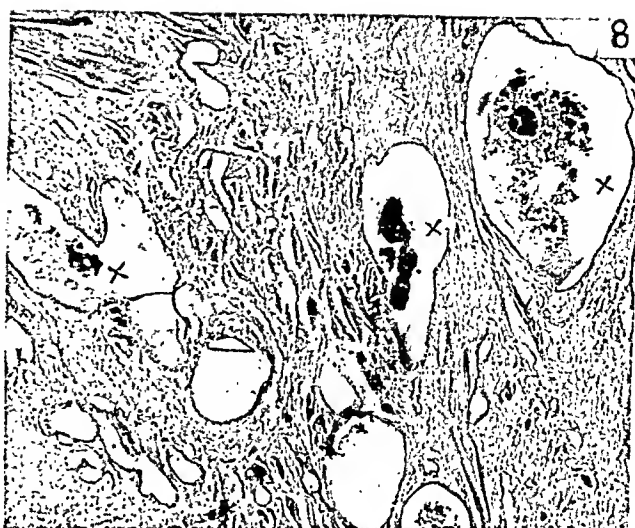
LEGENDS CONTAINED IN TEXT

The characteristic cyst of cystic disease or " chronic cystic mastitis," to use the older term, is lined by " pink " or " pale " epithelial cells, often described as sweat-gland in type. These small, dilating cysts later coalesce and form the large, blue-dome cyst of Bloodgood's description, which is characteristic of the advanced cystic condition. A large section of one of the breasts in a bilateral case, treated by double simple amputation, in a patient of 52, is shown in Fig. 3. The epithelium lining all these cysts was of so-called sweat-gland type and inactive or degenerating. These blue-dome cysts are, in my experience, essentially degenerative, though there may be considerable intracystic proliferation before the degenerative change sets in. I have been unable to find any evidence that these " pale " cells give rise to cancer, though other observers consider that they may be the starting-point of what is called " sweat-gland carcinoma of the breast." In Fig. 4 is shown a cystic breast of blue-dome type very similar to that of Fig. 3, but with a small early cancer (x) ; the genesis of this malignancy showed no " sweat-gland " character.

The other main type of degeneration in a duct is of a fatty character ; it produces cysts with proliferating epithelium breaking down into masses of colostrum-like, foam cells. Here again, the end-stage may be atrophy and quiescence, but in these fatty cysts there is a tendency for a residuum of intact cells to survive and persist in their proliferation, until a malignant cell type emerges. Such epitheliosis then becomes the genesis of cancer.

The enlarged " knobbly " cystic breast of the climacteric and menopausal period is, therefore, justifiably regarded with suspicion. Most, or even all, of the cysts may be of the blue-dome type and harmless because essentially atrophic ; others may be fatty cysts also eventually to become quiescent and atrophic. But partial cell degeneration in a fatty cyst or even fatty débris in a duct with little dilatation, may leave intact epithelial cells which can proliferate and which later become malignant. These possibilities explain the frequency of the question, what is the relation of cystic disease or "chronic mastitis" to cancer? They explain also the uncertain answer. For if the term chronic mastitis covers an identifiable clinical picture, it does not describe a defined pathological entity, but is a complex and variable lesion with different types and degrees of epithelial activity and degeneration. This varied histological picture of non-malignant menopausal tissue is seen in Fig. 5. It is not possible to say what the eventual outcome would have been in such cases, had the breast not been removed at this benign stage.

I have, of necessity, omitted many steps in my argument, but perhaps enough has been presented to show that the genesis of cancer in the breast can be traced to a persistent epithelial proliferation within the ducts. As adenosis may become adenoma (but not adeno-carcinoma), epitheliosis may become epithelioma, in the French use of this term, first within the duct, a duct (intraductal) carcinoma, which is the initial stage of

all mammary carcinoma and later, when rupture of the duct wall occurs, an infiltrating, disseminating, malignant growth. Malignant epitheliosis, at the intraductal, pre-invasive stage may form a cellular lining, or a solid filling or may be of papillary structure and it may develop in ducts showing all degrees of dilatation or in pre-formed cysts. This intraductal stage is shown in Fig. 6, with much central cell degeneration, the "comedo cancer" type ; this patient, after radical operation without irradiation, was alive and well 19 years later. The area illustrated shows a really early malignant tissue, possibly still at .the entirely intraductal stage. These are, unfortunately, rare cases of doubtful clinical import and are removed, as MacCarty of the Mayo Clinic noted, not because they are malignant, but because the surgeon fears they may be and histological examination then reveals the early cancer. After this early stage, prognosis deteriorates, for not only do malignant cells infiltrate the tissues of the mammary area, where they are accessible to therapeutic attack, but they enter the lymph stream and later, the blood stream and are thus transported to distant and usually untreatable sites. The stages of this spread are not rigid and cannot be traced for all cases, but they appear to occur more or less in the following sequence.

SPREAD

Initially, there is probably always some spread of the cancer cells up and down within the ducts themselves ; in some cases, there is clinical evidence of long-standing intraductal growth in cystic ducts, much of which was probably benign. Appearances do not, in general, suggest a unicentric, intraductal genesis, though single malignant papillomas occur. Very rarely, malignancy seems to emerge simultaneously over much, or even all, of the *corpus mammæ*, the ducts over a wide area showing comparable changes.

Invasion through the duct wall initiates the next stage, a spread largely influenced by the condition of the stroma and its lymph and blood vascularity. In old patients with advanced involution changes, invasive growth, usually slowly infiltrative, forms the so-called atrophic scirrhous cancer. In young patients and especially in the rare pregnancy and lactation cancer cases, facilities for rapid spread are afforded by a physiologically active and vascular tissue. These are the more unusual types. In general, after penetration of the duct wall, the cancer cells infiltrate the periductal stroma (Fig. 7, at x) and this may continue to a considerable extent before the tumour cells find their way into the lymph stream. Were this not so, histological evidence of malignant growth outside the ducts would be even more ominous than it is. The importance of the interval between a purely stroma infiltration and irruption into the lymph channels is indicated by the wide difference in survival rates between cases of cancer confined to the breast and those which show spread beyond the mammary tissues.

The invasion of the lymph stream in the mammary tissue indicates the next stage of spread ; it may be observed in an area of stroma infiltration or beyond it. Early, it is an embolic process, often well shown in lymph vessels of the sub-areolar plexus (Fig. 8) ; later, with slowing, blocking and diversion of the lymph stream, cancer spread becomes a permeation (Fig. 9). Mr. Sampson Handley has done the classic work on this type of malignant growth. The physiologist as well as the pathologist can learn much of lymph vessel anatomy and distribution from a study of these embolic and permeative processes. For the surgeon, the clinical implications are grave, as in a high proportion of cases invasion of the lymph stream in the mammary area implies invasion of the draining lymph nodes. I found this correlation in 88 per cent. in a series of over 600 mammary cancers treated by radical operation.

Invasion of lymph nodes, usually initially of the axilla, is seen in Fig.10, which also shows a malignant-cell embolus in an afferent lymph vessel (x). This is probably the early method of transport ; later, it becomes a continuous growth, with progressive destruction of lymphoid tissue and the possible diversion of malignant cells to unusual sites.

If tumour were restricted, at the time of treatment, to the breast and axillary nodes, radical operation for mammary cancer might have been, as Halsted hoped, one of the triumphs of surgery. But microscopical evidence of further extension may be found at the time when the patient is first examined and treated, such as invasion of the pectoral fascia and even of the underlying muscle, the axillary fat, the skin over the breast and, possibly, considerably beyond it. Some of these invaded areas are marked on the large section shown in Fig. 11. All such findings are indications of an advanced tumour condition. Only one remains to be considered, invasion of the blood stream; an appearance one finds with dismay and reports with reluctance, for it points to a blood-borne dissemination of cancer cells and the likelihood of widespread and inaccessible tumour deposits. Where there is definite evidence of bloodstream invasion to any degree, as in Fig. 12, with clinical demonstration of metastatic growth, the cancer has reached its final stages of spread, with the formation of distant and, in almost all cases, multiple malignant formations.

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THE TREATMENT OF PEPTIC ULCER BY VAGOTOMY

Condensed from a Hunterian Lecture delivered at The Royal College of Surgeons of England

on

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by

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MY INTENTION IN this lecture is to assess the position occupied by vagotomy in the surgical treatment of peptic ulcer, using as a basis for my conclusions the results of my own series of cases and the accumulated experience from the literature of this operation.

Ætiology

Causal Factors. Ulceration of the stomach and duodenum, as elsewhere in the body, is probably due primarily to trauma from the abrasive action of hard, sharp particles of food. This will obviously be accentuated by hurried, careless eating and by poor dentition. Another factor entering into the ætiology is that of sepsis. Böttcher[1] and Haines[2] first described the presence of organisms in the base and edges of peptic ulceration and their work has since been confirmed by Rosenow,[3,4,5] Sanford[3] and others. The organisms responsible are usually either diplococci or streptococci and the source of infection often lies in the naso-pharynx or teeth, the ulcer probably becoming infected through ingestion of mucopus. Other possible factors are arteriole spasm (von Bergman[6]), general toxæmia (Long,[7] Curling,[8] Perry and Shaw[9]), and possibly toxic neuritis (Pottinger[10]).

Ulcer Diathesis. When an acute ulcer develops from a combination of any of these factors, the natural healing processes of the body are opposed by the peristaltic movement and the gastric acidity. But Dragstedt[11] has shown that these lesions normally tend to heal spontaneously, despite this handicap, and it is possible that the intestinal mucus or the epithelial layer of Duran-Jorda[12,13] may afford the necessary protection. There is no doubt that the incidence of acute gastric ulceration is considerably higher than is generally recognised and the reason why chronic ulcer develops in particular instances must be sought in a consideration of the ulcer diathesis. The characteristics of this condition

have long been recognised and often described; but it is probable that the psychological type of the individual concerned is the essential factor and not the mere accident ,of physical build originally described by Goldthwait[14].

Experimental evidence. Cushing[15] first demonstrated the existence of the cortico-vagal tract and showed that irritative lesions of it gave rise to gastro-duodenal ulceration. Carlson[16] and Dragstedt[17] found that the psychic component of gastric secretion first described by Pavlov[18] was not limited to gastric digestion but present continuously, particularly in the ulcer diathesis. The work of Stahnke[19], Hartzell[20] and Beaver and Mann[21] showed that this secretion was due to stimulation of the vagus. Wolf and Wolff[22] demonstrated on the human subject that these effects can be produced by gastric hypersecretion secondary to psychological causes. It thus becomes apparent that the underlying cause in the development of chronic ulcer is hypersecretion resulting from -vagal stimulation.

Review of past physiological surgery. Many previous attempts have been made to deal with the subject of ulcer along physiological lines. Gastro-enterostomy, introduced by Wolfler in 1881, was the first of these. Its rationale was based on rest of the ulcer and a belief that the gastric acidity would be lowered by entrance of bile into the stomach. It proved a failure and surgical opinion became focused on subtotal gastrectomy as a possible alternative. This procedure, the most satisfactory up to the present time, depends on resection of the acid-producing portion of the stomach. The operative mortality is surprisingly low but the operation has certain disadvantages. The recurrence rate is at least 5 per cent. and quite a high proportion of patients are unable to return to full economic life. Even those who can do so tend to suffer from minor intestinal upsets, particularly if not sufficiently careful about their diet.

A considerable amount of experimental work has been performed in the last few years in an effort to discover alternative procedures without these disadvantages. The first of these was Wilson-Hey's attempt to reduce acid secretion after gastro-jejunostomy by ligation of the larger gastric vessels. A series of these operations was published by Somervell[23] and he claimed good results, but it seems doubtful whether they are really much better than are those of gastro-enterostomy alone.

The next attempt was an attack on the problem by gastro-duodenal denervation, which is the most rational line of approach on physiological grounds. Exner and Schwarzmann[24,25] performed the first sub-diaphragmatic section of the vagi but their results were not successful owing to incomplete neurectomy. Stierlin[26] Bircher[27] and Schiassi[28] elaborated alternative methods of vagotomy but none of these was sufficiently successful to achieve more than passing interest. (Fig. 1.)

PREVIOUS DENERVATION PROCEDURES.

4. nerves of posterior wall of stomach divided through multiple incisions in transverse mesocolon.
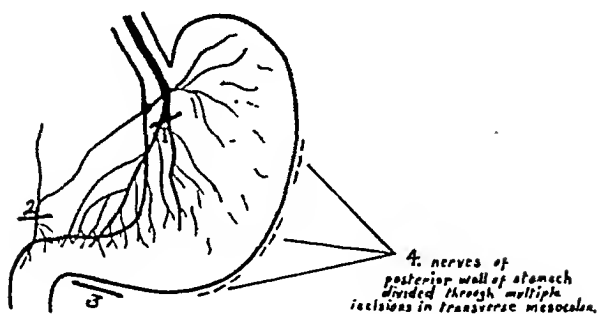
I. COMPLETE GASTRIC DENERVATION [LATARJET].

EXNER.

STIERLIN.

SCHIASSI.
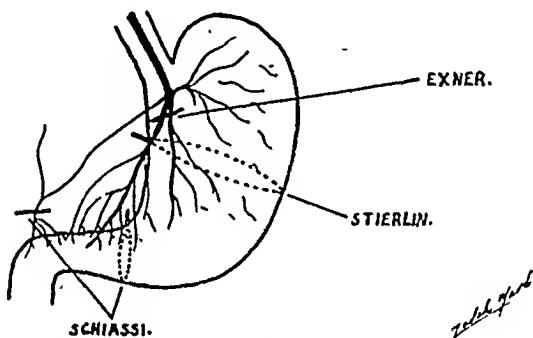
II. OTHER DENERVATION PROCEDURES.

Fig. 1. Previous Denervation Procedures.

Above. The original total gastric neurotomy of Latarjet, used with success in tabetic crisis.

Below. Past attempts at vagal denervation for duodenal ulcer.

Vagotomy

The most recent attack along these lines constitutes the modern operation of vagotomy. Hartzell[20] in 1929 reviewed the results of previous attempts at this procedure, all of which had been made through the abdominal route. Pieri and Tanferna[29] in 1930 first described the transthoracic approach, through which Dragstedt and Owens[30] reported several successes in 1943. The first abdominal vagotomy in its modern form was also performed by Dragstedt[31] in 1944. During the past four years a great deal of work has been done on the technique and afterresults of vagotomy, together with much detailed research into its physiological basis. This operation is no longer an experimental method of treatment and Table I shows the total number of large-series cases which have been operated on in Britain and America up to the present time.

TABLE I

LARGE-SERIES VAGOTOMIES
(since 1943)
U.S.A. and BRITAIN

DRAGSTEDT[31] (Chicago Univ.)	..	..	400	} personal communications
MOORE[57] (Harvard Univ.)	..	..	115	
GRIMSON[59] (Duke Univ.)	..	..	96	
WALTERS[61] (Mayo Clinic)	..	..	80	
CRILE[62]	..	..	77	
ORR[60] (Brit. Postgrad. School)	..	..	90	personal communication
Personal series ..	..	..	103	
TOTAL	..	..	961	

Selection of cases. Most surgeons working on vagotomy have confined it to the treatment of duodenal ulcer or the complications arising after gastrectomy and gastro-enterostomy. There is no doubt that it gives excellent results in these but I believe, contrary to Allen's[32] recent opinion, that it is also of considerable value in selected cases of gastric ulcer. It must, of course, be confined to the smaller, uncomplicated ulcers without too great a degree of surrounding induration, and in which there is no possible question of malignant change. My own series contains 30 such cases which have given very successful results, particularly in dealing with the otherwise refractory high lesions of the lesser curvature. A most important point is to avoid operating on patients with a marked psychotic element. Despite healing of the ulcer and reduction in gastric acidity, such cases will continue to show secondary psychological pain.

Anatomy and surgical approach. The best description of the detailed anatomy of the vagus is found in the work of McCrea, [33,34] Mitchell[35] and Kampmeier[36]. Each vagus divides posterior to the lung root into three or four main branches, which run downwards, partly on the œsophagus and partly in its substance, as an intricate nerve plexus. During its course through the thorax this plexus is joined by sympathetic filaments from the thoracic trunks and ganglia, and re-collects on each side just above the diaphragm into one or two main branches. These branches pass through the hiatus in company with the œsophagus and break up below it in the manner shown in Figs. 2 and 3.

There are therefore two possible modes of approach to the vagus—through the chest or through the abdomen. Early surgical work was done almost entirely by the trans-thoracic route, owing to the influence of Smithwick's[37] work on nerve regeneration. This led to the fear that unless long lengths of the nerve were excised the results would be unsatisfactory, but in actual fact the reverse was found to be true. The anatomical variations of the vagus in the thorax are so complex that it is extremely difficult to be certain of eradicating the entire nerve by an operation at this level. It is much easier to deal with it effectively at the

level of the diaphragm and the modern tendency is therefore towards the development of the trans-abdominal operation. I shall not attempt to deal in detail with the operative techniques of the two procedures. Those which I use personally are modifications of Dragstedt's original ones and will be described in a separate paper.

VAGUS SUPPLY TO STOMACH & DUODENUM.
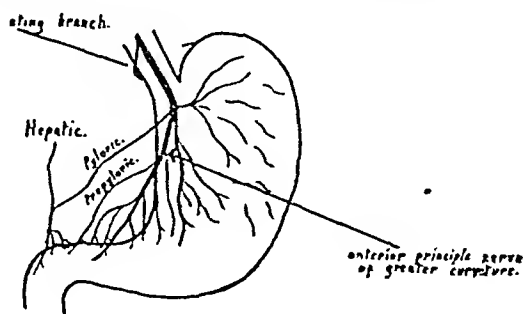
ANTERIOR VAGUS.
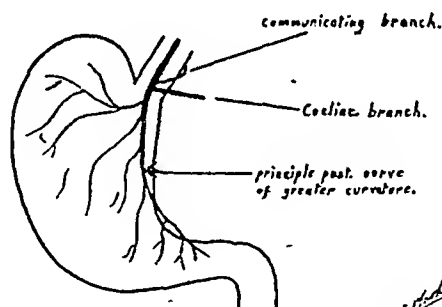
POST. VAGUS.

Figs. 2 and 3. Vagal supply of stomach and duodenum.

The Trans-Thoracic approach. This employs an approach through the left side of the chest by subperiosteal resection of the middle two-thirds of the 8th or 9th rib. Controlled-respiration anæsthesia is essential and lung expansion must be adequately secured before closing the pleural cavity. The operation is technically easier than the abdominal approach but suffers from certain disadvantages. Neurectomy is incomplete in more than 10 per cent. of cases and there is more risk to the patient, both from the added length of the procedure and the liability to chest complications. But the most serious drawback to its use lies in the fact that the abdominal lesion cannot be inspected, and this has sometimes led to grave consequences. The after treatment of these cases is along the

well-established lines of thoracic surgery. Personally, I believe that this approach is very rarely indicated.

The Trans-abdominal approach. This is the one which I now employ routinely. The usual pre-operative investigations are carried out in the Out-patient Department before admission and the chest is always carefully examined, since the high abdominal incision and the interference with the diaphragm at operation combine to favour the development of post-anæsthetic complications. Another important test at this stage is the estimation of the night fasting juice, which is carried out by means of continuous gastric suction overnight, while the patient is asleep. Comparison of the acid levels of the night fasting juice before and after vagotomy forms one of the most valuable tests of the efficacy of this procedure.

The incision employed is a high right paramedian one and mobilisation of the left lobe of the liver is usually necessary before an adequate view can be obtained. The œsophagus is mobilised after incising the peritoneum over it transversely and the vagal branches then identified and secured. It is of the highest importance not to miss any of them, since failure to secure complete neurectomy means failure of the entire procedure. The technique is more difficult than is that of the trans-thoracic approach but, when accustomed to it, the whole operation takes less than half an hour. The secret of success lies in careful positioning of the patient, adequate lighting and experienced assistance. Immediate post-operative treatment consists of continuous gastric suction until the temporarily paralysed stomach recovers its power of movement. Intravenous feeding is employed during this period and breathing exercises are instituted after the first few hours. As soon as the suction is discontinued, a rapidly increasing diet is given and patients are discharged from hospital on a modified ulcer diet, with instructions to remain on it until later review. I feel that it is dangerous to allow them to return to normal diet until there is definite evidence that the ulcer has healed.

At the end of three months all cases are again reviewed. The barium meal is repeated and almost always shows complete healing of the lesion. Another fractional test meal is performed and the night fasting juice again investigated. An insulin test meal is also carried out on every case by the technique described by Hollander.[38] The rationale of this test depends on the fact, first demonstrated by Roholm,[39] that excessive gastric secretion takes place during hypoglycæmia. Okada[40] and La Barre[41] showed that this secretion is abolished following vagotomy or after administration of atropine, and the insulin test meal therefore supplies conclusive proof whether vagotomy has been complete. In the normal person or when neurectomy has been incomplete, the response to insulin hypoglycæmia is very marked, whereas it is almost non-existent following complete vagotomy. (Fig. 4.)

THE INSULIN TEST MEAL CURVE
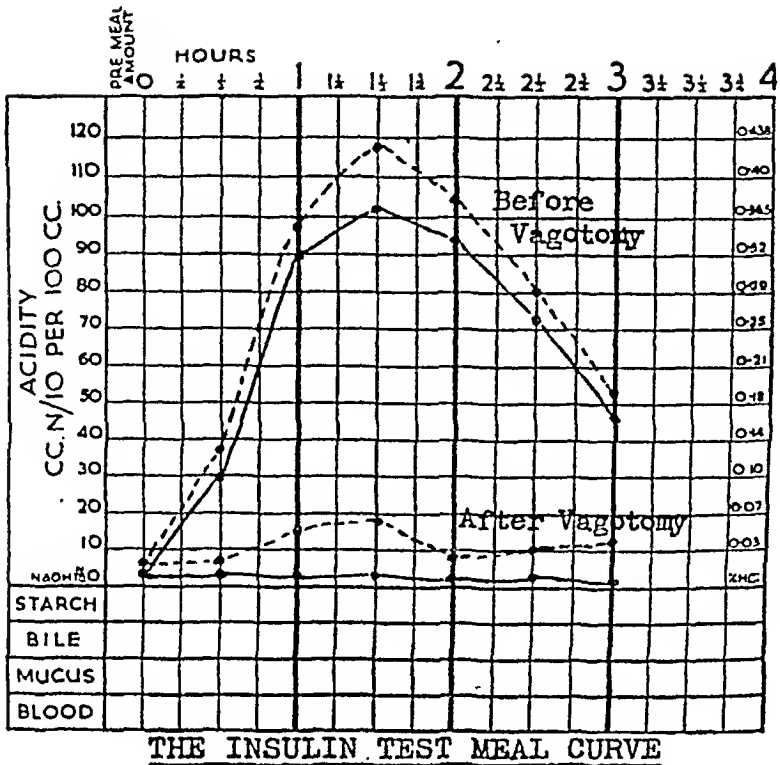
Fig. 4.

Results of Vagotomy

Physiological Sequelæ. The physiological sequelæ of vagotomy may be divided into immediate and delayed effects. The immediate effects are temporary paralysis of the stomach and complete relief of ulcer pain. The gastric paralysis was first described by Klee[42,43,44,45], and Exner and Schwarzmann, [24,25] and is the natural result of division of the main motor nerves. Carlson[46] and McCrea[33,34] found during their investigations into the functions of the gastric nerves that the vagus is solely responsible for the control of peristalsis, and that the sympathetic is not an opponent nerve supply but responsible for maintenance of muscle tone. The relief of pain following vagotomy, which is so marked a feature, is probably due to cessation of the irritating effect of strong peristaltic contractions and muscle spasm. Bentley and Smithwick's work[47] suggests that the sympathetic is also the main pathway for sensory stimuli, and Hesser's [48] and Moore's [49] investigations on the human subject confirm this to be the case as regards the stomach and duodenum.

It is because of the temporary paralysis that post-operative gastric suction is necessary. Table II shows the period for which it was required in my series of cases and the average quantity of fluid withdrawn. Despite

the variation in vagal tone pre-operatively, it is interesting that there is no essential difference between the degree of immediate paralysis in gastric or duodenal ulcer. Also that the severity of this paralysis apparently bears little relationship to the extent of the later retention symptoms.

TABLE II

. POST-OPERATIVE GASTRIC SUCTION

PERIOD REQUIRED

58 per cent. required suction for 36 hours or more
(average period 32·6 hours)

VOLUME OF FLUID WITHDRAWN

100 c.c. or less .. .. 38 per cent.
2,000 c.c. or more .. .. 10 per cent.
(average volume=610 c.c.)

NOTES :—
1. No apparent difference between types of ulcer.
2. No similarity between degree of post-operative paralysis and later retention symptoms.

The delayed effects of vagal section are of equal importance. Recovery from the temporary paralysis takes place within 24-72 hours and peristalsis gradually increases in frequency and amplitude, though it probably never returns to the previous extent. Moore[49] and Hesser and Baylin[50] have investigated this subject extensively. Most of my own cases showed considerable delay in gastric emptying three months post-operatively, to which I shall refer again later. Associated with the gradual return of peristaltic activity there is an increase in muscle tone, which Hesser and Baylin[50] have shown may reach 2-3 times its pre-operative strength within three years. Curiously enough this is often accompanied by temporary gastric dilatation and ptosis, the result of pyloric spasm from the unopposed action of the sympathetic, and it may be some months before the stomach regains its normal size and position. Rundles and Baylin[51] have shown that these alterations in muscle tone and movements are not confined to the stomach but also involve the duodenum and jejunum ; but according to Moore[49] no other organs supplied by the vagus appear to show any alteration in function. Dr. Forbes Lawson[52] informs me that there is radiological evidence of pylorospasm after vagotomy and Moore[49] has shown that the initial emptying time is often considerably lengthened.

Both the total volume and free acidity of the gastric secretion are permanently reduced after vagotomy. This was first described by Hartzell[20] in connection with the fractional test meal and by Thornton[53] and Grimson[54] as regards the night fasting juice. My own work confirms these observations and shows the free hydrochloric acid secreted during the fractional test meal to be reduced to one-third of the pre-operative quantity. (Table III, Figs. 5, 6.)

TABLE III

NIGHT FASTING JUICE

Average result in 40 cases of duodenal ulcer.

BEFORE VAGOTOMY

		c.c.s. per hour
Total secretion	..	67·3
Total acidity (as N/10 NaOH)	..	49·7
Free HCl (as N/10 NaOH)	..	32·2

AFTER VAGOTOMY

Total secretion	..	37·0
Total acidity (as N/10 NaOH)	..	24·8
Free HCl (as N/10 NaOH)	..	4·7
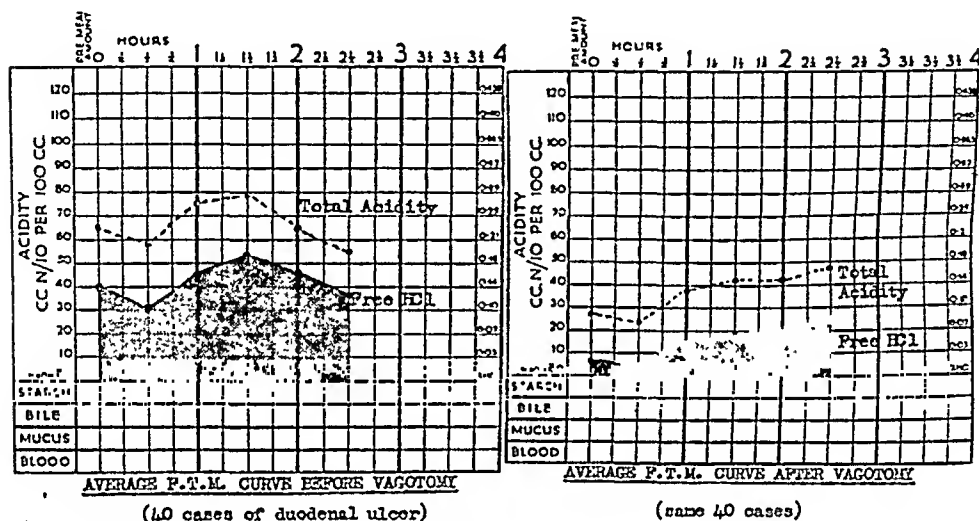
Figs. 5 and 6.

Pfeiffer[55] demonstrated in dogs that there is a temporary drop in blood calcium and Moore[49] has confirmed that this also occurs in man. The fall is analagous to that occurring in gastric tetany and is due, I think, to the retention which takes place.

Personal results. In company with other surgeons performing vagotomy I find it to give excellent results in properly selected cases. Two of my earlier trans-thoracic operations gave evidence of incomplete neurectomy at subsequent insulin test meal. Both patients continued to experience ulcer symptoms and were subsequently re-operated on by the abdominal approach. In each a large vagal trunk was found to be present, and division of this gave complete and lasting relief. With these exceptions, every case showed entire disappearance of ulcer symptoms and radiological evidence of healing within four months. There has been a rapid increase in weight, which compares favourably with the usually slow regain after gastrectomy, but the most dramatic feature is the psychological reaction of the patient after operation. They are all very enthusiastic and over 60 per cent. of them returned voluntarily to work before coming up for their routine post-operative review.

One death occurred in my series of cases, from hæmorrhage ten days after operation. The cause of this was found at post mortem to be a large aneurysmal dilatation of the right gastric artery, immediately underlying the ulcer crater, which must have ruptured when peristalsis recommenced. During his five-year history of ulcer symptoms there had been no suggestion of previous hæmorrhages in this patient. The operative mortality in uncomplicated cases has been found to be extremely low in all the published series of figures. The only serious post-operative sequela has been the occasional development of chest complications due to the high incision and, I believe, to the combination of cyclopropane and curare in this operation. Since substituting ether for cyclopropane, I have had less trouble in this respect.

The only unpleasant symptoms after operation in my series have been those due to temporary gastric retention. The majority of cases show at least a six-hour delay four months afterwards, although this has usually disappeared by the end of the first year. Associated with it is a feeling of distension after food and some degree of foul eructation, no doubt due to fermentation in the delayed stomach contents. But these symptoms have only been of major importance in relatively few cases, and Table IV shows their incidence in my own series. In an attempt to prevent them, some of the American surgeons have combined gastro-enterostomy with vagotomy but this has not been generally successful. Minor retention symptoms often seem to continue, despite the presence of the anastamosis. Dragstedt[56] has recently suggested that the chief cause of prolonged retention symptoms is overstretching of the stomach musculature due to insufficiently prolonged post-operative suction. The resultant paralytic dilatation is only slowly overcome by the gradually increasing stomach tone and may not be completely corrected for several months.

TABLE IV

RETENTION SYMPTOMS AFTER VAGOTOMY
(up to four months post-operatively)

RADIOLOGICAL
　　88 per cent. showed　6-hour gastric delay
　　30 per cent.　　,,　　24-hour　　,,　　,,

SYMPTOMATIC
　　74 per cent. showed no significant symptoms
　　22 per cent.　　,,　　moderate symptoms
　　4 per cent.　　,,　　major, severe symptoms

Associated with this retention phenomenon is the presence of intermittent diarrhœa, which Dragstedt[56] thinks is probably due to the associated gastric fermentation. Another curious sequela which occurred temporarily in two of my cases was a typical " dumping " syndrome. The fact that this took place in patients showing an unusually severe degree of retention is rather surprising in view of the modern theory of the causation of this condition, and casts some doubt on the accepted

hypothesis. Although these retention symptoms disappear within a few months of operation, Dragstedt[31] has found that they tend to recur at intervals throughout the whole follow-up period, in a milder form. He has noticed that they coincide with the same psychological conditions which would normally have produced ulcer pain. This interesting observation suggests that the nervous impulses consequent on psychological stress, unable to use the abdominal vagus channel as an outlet, overflow into the corresponding portion of the sympathetic system.

Another occasional post-operative complication which has been described is temporary dysphagia, though I have not experienced it in my own series of cases. This condition is thought to be due to the alteration in enervation of the lower end of the œsophagus and cardiac orifice and, possibly, to the trauma inflicted on these structures at operation. It has never been a sufficiently serious disability to warrant corrective measures, and usually disappears spontaneously after a few weeks.

In an attempt to overcome both the immediate retentive symptoms and these later ones, a considerable amount of work has been done in America on parasympathetic stimulants. The latest and most successful of these is " Urecholine." This preparation is not yet on the market and is still in its experimental stages, but it does appear to give considerable relief. It seems probable that further development along these lines will eventually remove the only unpleasant sequel to vagotomy.

I have had no case of ulcer recurrence following operation, although this has been described by others. Moore[57] gives a recurrence rate as high as 5 per cent. but it is possible that incomplete vagotomy may account for some of his failures, since he always uses the trans-thoracic approach. This suggestion is confirmed by the experience of Dragstedt,[58] who operated a second time on half his own failures from the trans-thoracic procedure and found evidence of incomplete neurectomy in all of them. No proven recurrences have yet been reported following operation by the abdominal approach.

Conclusions

In conclusion, I am convinced that vagotomy constitutes a great surgical advance in the treatment of peptic ulceration. It is not the final solution, for I believe that control of the ulcer diathesis is essentially a medical and an economic problem. It does not seem beyond the limits of reasonable possibility that suitable drugs will eventually be developed to control the vagotonia which is the cause of the ulcer diathesis. But until that stage is reached, surgical treatment of chronic peptic ulcer must continue to be the method of choice, with vagotomy as the most successful approach in selected cases.

You will notice that I have not referred at all in this lecture to the end results of vagotomy. Dragstedt's original cases have now been followed

up for over five years and are still giving satisfactory results. But with the lesson of gastro-enterostomy before us, it would be foolish to assume permanency of cure until a large number of cases have been carefully followed for at least 10 years.

I should like to end on another note of warning. Over-enthusiasm in any recent surgical advance is apt to produce unnecessary failures, which detract from their true value, and it is most important that this should not take place in vagotomy. Every case must be checked post-operatively to ensure that neurectomy has been complete, since it is only in the light of this knowledge that it is possible to assess the ultimate results of the operation.

Finally I should like to acknowledge the kindness of Professor Dragstedt, Dr. Grimson and Dr. Moore, for the help they have given me out of their larger experience. I am indebted to them and to Mr. Ian Orr for permission to use their results and conclusions in this paper; to Dr. Forbes Lawson for his radiological examinations of my cases; and to Dr. Wale for the pathological investigations on them. To the nursing staff of the Leicester City General Hospital a particular tribute is due. Without their willing co-operation and untiring care, it would have been impossible to attain the successful results that have been achieved.

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Fellows and Members of the College.

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There is an inclusive charge of £1 5s. (including drinks), which must be sent with the application to the Assistant Secretary at least a week before the date of the dinner. The dress is Lounge Suit or Uniform.

THE ENDAURAL APPROACH TO THE
MIDDLE AND INNER EAR

by

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THERE HAVE BEEN many attempts to devise for operative procedures on the ear a surgical technique which did not involve a post-auricular incision. These have often had a limited application in that the patients must be carefully selected and they have mostly involved operating through some sort of speculum, e.g., Prof. V. Eicken in Berlin did an attico antrotomy with an incision along the roof of meatus, operating with a duck-billed speculum. Both of these factors are serious disadvantages and it was not until Dr. Lempert of New York published his paper (Arch. of Otol. May, 1938, Vol. 27, p. 555-587) that an alternative method of approach to the mastoid and middle ear, applicable to all cases, was generally known.

I saw Dr. Lempert operate in this manner in 1938 and was amazed at the exposure obtained through this incision. One cannot adequately visualize the extent of the exposure from reading a description of the operation, and it is only by seeing and doing the operation that one is convinced that this incision provides an adequate exposure for carrying out any bone surgery indicated in the ordinary case for which a mastoid operation is performed. Since then many surgeons have used this technique and in general they consider the advantages outweigh the disadvantages for a modified or classical radical mastoid operation, but I should say that comparatively few surgeons in this country have adopted the endaural approach for a simple mastoidectomy as their routine operation. I, personally, have ceased to use it for the simple mastoidectomy and will give my reasons later for first adopting it and then discarding it.

Since mastoid surgery was begun the post-auricular incision has been the standard method of approach and has given good results, but it has always caused dissatisfaction and we must consider in detail the reasons for this. The simple mastoidectomy for acute suppuration in the mastoid process will be considered first. The objects of the operation are twofold : first, complete exenteration of all the mastoid aircells, including the marginal, tip, retrofacial, perilabyrinthine and zygomatic groups ; and secondly, obliteration of the resultant cavity. Originally the operation for acute mastoiditis limited its objective to providing adequate drainage for the mastoid antrum, but many disappointments in the form of post-operative intracranial complications and recurrent post-auricular abscesses

gradually widened the scope of the operation until, by the 1920's, largely as the result of the teaching of Jenkins in this country and Neumann in Vienna, it was recognized generally that a simple mastoidectomy should aim at the complete exenteration of all mastoid aircells, with the exception of the apical petrous cells and the peritubal cells. This naturally left a large operative cavity. If this was allowed to heal by granulation the convalescence was slow and many painful dressings were required. It was no unusual thing for a mastoid to take three months to heal. The end result was an ugly, depressed post-auricular scar covered by atrophic skin. Many attempts were made to overcome this by partial suture, secondary suture and complete suture with healing by blood clot. All these methods entailed some risk and were condemned by Neumann in 1926 in a paper read to the B.M.A. This was still the position in 1938 when Lempert introduced his endaural approach. He claimed that through this approach the mastoid cells could be completely exenterated and that without painful dressings and without an ugly post-auricular scar the resultant cavity could be obliterated. This claim was justified, in my opinion, and a complicated infected cellular system in the mastoid could be adequately dealt with through this incision. The operation itself, is, however, rather tedious as compared with the wider exposure of the post-auricular incision and takes perhaps half as long again. The advent of sulphonamides and penicillin have made it safe and satisfactory to insufflate the cavity with a post-auricular incision, with a mixed powder and then sew up the wound with a rubber drain for the first 48 hours. Painful dressings are thus eliminated and the wound may be expected to be healed in 14-21 days. Thus the main advantage of the endaural approach for a simple mastoid is negatived, and, in my view, at the moment there is no adequate compensation for the increased tediousness of the operation through the endaural approach.

The radical mastoid where surgery of the middle ear and attic is to be performed is a different problem and in my opinion the endaural incision is the method of choice. The reasons for this are that (1) There are satisfactory landmarks in sight at all stages of the operation. (2) The surgeon looks straight at the middle ear instead of obliquely. (3) Chronic otitis media is a disease of the patient with a non-pneumatised mastoid and the only reason for removal of the mastoid bone is to provide access to the mastoid antrum. The endaural approach allows adequate exposure with removal of less bone. (4) There is no post-auricular wound to give trouble. (5) The muscles attached to the mastoid tip are not disturbed. (6) A satisfactory meatus is obtained without external deformity. (7) Bleeding is more easily controlled. (8) A forward lateral sinus does not increase the difficulty of the operation.

Of these reasons I consider number one—the visualizing of satisfactory landmarks at all stages of the operation—to be the most important. It is also noticeable that there is appreciably less hæmorrhage with the endaural approach than with the post-auricular incision. Before the middle

ear can be well seen in the classical radical with the post-auricular incision, the meatal flap must be cut. This is most conveniently done when the bridge has been taken down, and it always causes hæmorrhage. So that at the moment when the surgeon wants to see clearly in the middle ear he causes hæmorrhage which is difficult to control. The endaural approach avoids this trouble by making the meatal incision at the beginning of the operation and by the time he comes to careful inspection of the middle ear the hæmorrhage has ceased.

Operative Technique

The incision employed is extra cartilaginous, and adequate exposure is obtained by virtue of the gap existing between the tragus and the helix.

The incision varies according to the type of operation proposed. For a simple mastoidectomy the bony meatus is not encroached on, and for a radical, or modified radical mastoid, the incision is carried down to the tympanic ring. In both cases a triple incision is made outlining a triangular piece of the posterior meatal wall. All these incisions are carried down to the bone. In a simple mastoidectomy the first incision is made on the roof of the meatus beginning internally at the outer part of the bony meatus. This incision is made outwards into the gap between the tragus and helix and carried upwards in front of the helix. The second incision starts at the same point on the roof of the meatus and is carried obliquely outwards across the posterior meatal wall to reach the floor of the meatus and stopping short of the cartilage. The third incision joins these two and keeps just in front of the free border of the cartilage of the fossa antitragica. These three incisions are now carried down to the bone and a triangular piece of skin, subcutaneous tissue and periosteum is thus cut free from the surrounding tissues. This is now freed from the bone with a periosteal elevator and thrown away.

Working through this window with a periosteal elevator the periosteum is elevated over the whole mastoid process and this elevation is carried up into the temporal fossa and forward onto the root of the zygoma. In this way the ear is mobilized and the window can be moved to expose any part of the mastoid process. Mollison's self-retaining mastoid retractor, or hand-held rake retractors, are inserted to give exposure.

The radical mastoid requires excision of a large piece of skin and periosteum as the bony meatal wall is going to be excised. The three-sided incision starts at the tympanic ring on the roof of the meatus instead of at the outer margin of the bony meatus as is done in the simple mastoid. The second incision starts at the same point on the tympanic ring, and the third joins the outer ends of the first two running just in front of the anterior border of the cartilage in the fossa antitragica.

When this incision is being made it is clear that comparatively little bleeding will interfere seriously with the visibility, and I have found it advisable to take steps to avoid this. The area should be well infiltrated with ½ per cent. Novocaine to which two minims of one in 1,000 Adrenalin

for each 10 c.c. have been added. This infiltration should be done twenty minutes or more before the incision is made, though even if done immediately before it is comparatively effective. The solution should be used freely for subperiosteal infiltration over the whole mastoid and a few c.c.s should be injected between the tragus and the helix. A suction apparatus used during the operation will keep the field clear and vision unimpeded.

It is clear from consideration of this exposure that the surgeon who is accustomed to work with a post-auricular incision will have to modify his bone technique, as it is not possible to slope the gouge and bevel the edges of his bony excavation when working through this incision. In the simple mastoidectomy the cortex is taken off with gouges in the region of the supra meatal triangle and the infected cellular system is then broken down with curettes and the cortical bone removed with bone forceps. In this way the tip and the angular cells can be easily and completely dealt with. The zygomatic cells are right under the incision and can be dealt with either by gouges or curettes and bone forceps. The Mollison self-retaining retractor is very satisfactory for most of the operation, but for the tip cells and marginal cells it should be replaced by hand retractors which give greater mobility to the incision and allow it to be displaced backwards and downwards to expose the tip.

In the radical mastoid Lempert recommends and practises opening the antrum through the postero-superior meatal wall with a dental drill. The drill is applied at the junction of the inner third and outer two thirds of an imaginary line along the postero-superior bony meatal wall joining the spine of Henle with the junction of the tympanic ring and tympanic membrane. The antrum is 1 to 2 mm. below the surface at this point in an inwards and backwards direction. When the antrum is opened the hole is enlarged in an outward direction with coarse burrs and the antrum exposed in this way. I have used this technique but have given it up as it means extra equipment, a dental drill and motor in the theatre, and the operation can be performed perfectly satisfactorily with the standard equipment. When the incision has been made and the meatal flap excised the surgeon gets a clear view at one and the same time of the supra meatal triangle, the bony posterior meatal wall and the tympanic membrane. Using gouges the bone removal is begun at the spine of Henle and proceeds forwards and inwards until the antrum is exposed, taking the bony posterior meatal wall down as the excavation proceeds. This is an entirely safe procedure as the landmarks of the middle ear are clearly in view all the time, and the relative positions of the antrum and the bone excavations are constantly checked. With the post-auricular incision it is advisable to leave the meatal wall standing until the antrum is opened, as it is the only reliable landmark if the middle ear is not in view. Taking the meatal wall down in this way expedites the antrum exposure very considerably and minimises the inconvenience caused by a forward lateral sinus and a low dura of the middle fossa. The forward

sinus is no longer a bogy with this incision, because one is working from before backwards, but the low dura of the middle fossa is still an inconvenience. When the antrum is well opened the bridge can be taken down under complete vision, as the middle ear side of the bridge can be as clearly seen as the antrum side of the bridge. At this stage two great advantages of this approach become evident. First, when the middle ear and attic are exposed by removal of the bridge it is important to have a bloodless field so as to see the pathological condition of the attic. This is obtained as there is no recent meatal incision to bleed into the wound, the meatal flap having been excised as the first step in the operation. In the radical with a post-auricular incision the flap is usually cut just before or just after taking down the bridge and the bleeding is always troublesome. Secondly, a direct view of the attic and middle ear is obtained instead of an oblique view from behind. I have been very impressed with how close one appears to be to the attic with this approach and how easy it is to see and deal with the pathology in the attic and the middle ear.

The post-operative treatment is simple in both types of operation. At the end of operation the pre-auricular extension of the incision is closed with one or two stitches. In the simple mastoidectomy the cavity is insufflated with penicillin and sulphathiazole powder and a rolled tulle gras wick is inserted through the defect in the skin of the posterior meatal wall. This serves to prevent the opening from becoming blocked with blood clot. A pad is placed behind the ear to depress the soft tissues into the bony defect. In 48 hours the tulle gras is removed and the cavity can be insufflated through a large aural speculum. The meatal wound is kept open for regular inspection by insertion of a speculum and, if necessary, by cauterisation of the edges with silver nitrate until the cavity is obliterated by granulations. When the cavity is obliterated the meatal wall heals soundly with no deformity. Narrowing of the meatus is not a complication which can be expected after this operation. This operation thus fulfils two most important desiderata of the simple mastoid operation in that it provides constant drainage for the cavity and allows the cavity to be inspected until it is obliterated by granulation tissue. I keep a dressing on the ear until about the seventh day, after which the patient keeps sterile cotton wool in the meatus until the wound is healed, i.e., three to four weeks. In 1938 when I began to use this approach it provided definite advantages over the post-auricular approach with regard to after-treatment as I did not like sewing up the wound in cases of acute mastoiditis. However, the use of penicillin and sulphathiazole powder for dusting the wound at the end of operation has so altered the post-operative course that it is now satisfactory to sew up the wound in acute mastoiditis with only a small drain for the first 24 hours that I now use the post-auricular incision for most cases of acute mastoiditis because of the greater technical ease of the operation with the post-auricular incision. With an extensively pneumatised mastoid there is no doubt that the

mastoid operation is more tedious with the endaural incision and takes me longer to complete, though the operation can be thoroughly performed in this way. With the present technique of after-treatment of the post-auricular wound there are thus no painful dressings and the main advantage of the endaural incision no longer exists. I sometimes wonder whether the present technique of dusting the cavity with penicillin and sulphathiazole and suturing the wound is not going to lead to an increase in the number of post-auricular abscesses as a result of failure to obliterate the mastoid cavity. I have seen several of these in the last year or two and if this does occur then I will revert to the endaural incision for simple mastoidectomy.

In radical mastoids the after-treatment is the same as with the post auricular incision, except that there is no post-auricular wound to consider. This absence of the post-auricular wound can only be an advantage. In addition there is no disturbance of the muscles attached to the mastoid process as it is most unusual in cases requiring a radical mastoid operation to find more than a few cells round the mastoid antrum. Thus the operation causes no limitation of head movements and the patient has very little discomfort at all in the post-operative period. It is my habit to complete the operation by insufflating the cavity with penicillin and sulphathiazole powder and then to pack the cavity with a quarter inch wide strip of rubber cut from the wrist of surgical rubber gloves. This rubber is dusted with the same powder while it is being packed into the cavity. I have copied this technique from Mr. Cann of Guy's Hospital and it is a most comfortable and satisfactory dressing. It does not stick, it can be removed painlessly and its removal does not make the cavity bleed. The rubber is removed on the fifth to seventh day and the cavity is mopped out and insufflated with powder daily after its removal. Epithelialisation is usually rapid and it is unusual to get more than a scanty serous discharge at any time.

In my opinion the endaural approach has very definite advantages for the radical or modified radical mastoid operation and is the operation of choice.

With this approach one has come to expect, and usually gets, a bloodless field and this makes it possible to refine one's surgical technique. I now rarely do a classical radical mastoid. Instead, in the great majority of cases, I preserve what remains of the tympanic membrane. Clearance of the anterior attic recess, tucked away anterior to the head of the malleus is an essential part of the operation and so it is necessary to excise the head of the malleus. This can easily be done with strong stitch scissors if the anterior part of the outer attic wall is first removed. In general, the failure to get the scissors on to the neck of the malleus is a sign that the outer attic wall is not properly removed.

It is my view that the increase in deafness after a radical mastoid operation is due to fibrosis on the internal wall of the middle ear around the oval and round windows. It is reasonable to suppose that preservation

of the tympanic membrane will reduce this, and it is for this reason that I employ the modified radical procedure particularly in those cases where the hearing is good before operation, e.g., attic erosion with cholesteatoma in the attic.

The use of the endaural approach for the fenestration operation has been firmly established by Lempert (Arch. of Laryng., Vol. 34, Nov., 1941). Since then he and others have introduced many modifications but in general the endaural approach is used for this operation. The technique of this operation is highly specialised and requires much practice. It cannot be learnt by reading and I do not propose therefore to repeat the steps of the operation. The results are encouraging and over 50 per cent. of the patients subjected to operation may expect lasting improvement of hearing, but it is still impossible so to select cases that the failures are eliminated even when the surgical technique is impeccable.

ROYAL COLLEGE OF SURGEONS OF ENGLAND
FACULTY OF ANÆSTHETISTS

AT THE REQUEST of the Association of Anæsthetists of Great Britain and Ireland, the Royal College, acting under the powers granted in the new Supplemental Charter has formed a Faculty within the College for those devoting themselves to the science and practice of anæsthetics.

Dr. A. D. Marston and Dr. Bernard Johnson have been elected Dean and Vice-Dean of the Faculty respectively.

The Faculty is governed by a Board, the members of which are as follows :—

DR. A. D. MARSTON, *Dean.*

DR. BERNARD JOHNSON, *Vice-Dean.*

DR. I. W. MAGILL, DR. E. S. ROWBOTHAM, DR. C. LANGTON HEWER, DR. R. E. PLEASANCE, DR. W. ALEXANDER LOW, DR. FRANKIS T. EVANS, DR. JOHN GILLIES, PROFESSOR R. R. MACINTOSH, DR. J. H. CHALLIS, DR. GEORGE EDWARDS, DR. KATHARINE G. LLOYD-WILLIAMS, DR. B. L. S. MURTAGH, DR. A. H. MUSGROVE, DR. H. J. BRENNAN, DR. VERNON F. HALL, DR. RONALD FRANCIS WOOLMER, DR. G. S. W. ORGANE, DT. T. C. GRAY, DR. E. A. PASK.

SIR ALFRED WEBB-JOHNSON, Bt.,
    K.C.V.O., C.B.E., D.S.O., T.D. } *President*

SIR CECIL WAKELEY, K.B.E., C.B.   } *ex officio.*
MR. L. E. C. NORBURY, O.B.E.   } *Vice-Presidents*

The Board held its first meeting on Wednesday, 24th March, at which future policy was discussed. It was decided to grant a Fellowship of the Faculty (F.F.A., R.C.S.) to those achieving distinction in the specialty.

A dinner was held in the evening to celebrate the founding of the Faculty.

"OBSERVABLES" AT THE ROYAL COLLEGE OF SURGEONS

5. JOHN HUNTER'S TANKARD

ONE OF THE MOST prized pieces of plate possessed by the College is John Hunter's tankard. It is a silver tankard with lid, and stands seven inches in height. It is of fluted design, bears the hall-mark of George II 1738, and was made in London by George Wickes.

Inside the lid a silver plate has been fixed bearing the inscription :— " This Tankard, the property of John Hunter, was presented to the Royal College of Surgeons of England by his nephew—Captain Sir Everard Home, Bart., R.N."

On a shield in the design on the front of the tankard is a Coat of Arms—on one half, three dogs of chace and on the other, two hunting horns stringed, and, for a crest, a small tree.

The design differs from the Coat of Arms granted to William Hunter by Lyon, King of Arms, in 1752, to which of course John had no right, but like William's Arms it includes dogs of chace and hunting horns. The design also differs from the Arms on John Hunter's coffin, which could not be defined with certainty, but were probably identical with those on the Memorial Brass in Westminster Abbey—three dogs of chace, two and one, and a hunting horn (for Hunter) on one half of the shield ; and on the other a lion rampant between two piles, with a bordure charged with six popinjays of the field (for Home—John Hunter's wife's family). In a crest on the memorial brass is an arm embowed in armour, holding an arrow.

The vexed question of John Hunter's right to the use of Arms is fully discussed in " A Memoir of William and John Hunter " by George C. Peachey.

<div align="right">A. W-J.</div>

SAYINGS OF THE GREAT

" The reception of new ideas tends always to be grudging or hostile."—
<div align="right">*Trotter.*</div>

" ' Old friends are best ' we lightly say
But as they pass upon the way,
Keep full the ranks with newer friends
Till time the adjective amends."—*S. Weir Mitchell.*

" Instead of herds of oxen, endeavour to assemble flocks of friends about your house."—*Epictetus.*

John Hunter's Tankard.
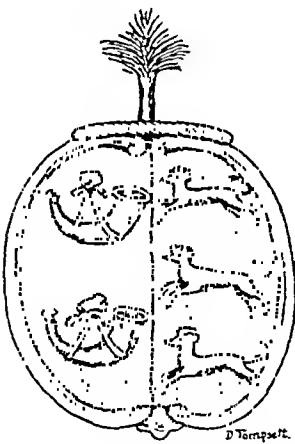
Arms from John Hunter's Tankard.
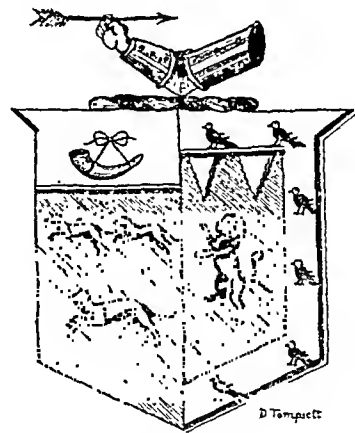
Arms from John Hunter's coffin
plate and Memorial Brass.

THE LIBRARY

CATALOGUES AND LOCATION OF BOOKS

IN A PREVIOUS article[1] the College's collection of periodicals was described, and some account was given of the principal helps towards their use which are available to readers in the Library through various catalogues and indexes.

While periodicals form the growing edge of the library and are numerically the most valuable part of the collections, the provision of separately published books is no less important a function of the library. It may be helpful to readers and students to describe the arrangement of the various groups of books and the catalogues which record them.

The Library acquires all important new British books in Anatomy and Surgery and the related specialties such as Anæsthesia, Orthopædics and Urology, with a selection of American publications and the more important French books. The work of other countries and in other languages is well represented among the periodicals, and separate publications are only obtained in very special cases; in the great period of German science, through the nineteenth century, German books were more plentifully obtained than those of any other foreign country. The Library covers in the same way the literature of Dentistry, Ophthalmology, general Pathology, Physiology and to a lesser extent Cardiology, Endocrinology and Neurology in their anatomical and physiological aspects. General medicine is adequately represented, but without attempt to cover clinical medicine in detail, and only those medical sciences are provided for which are of direct value in surgery, such as Biochemistry, Pharmacology and Radiology.

The modern books—those published in the last twenty-five years—are housed in the Erasmus Wilson room, which forms an open extension of the Reading Room. These modern books, both textbooks of general subjects and monographs on special topics, are grouped under large subject-headings, or classes, such as those named above. These classes are arranged in alphabetical sequence round the wall-cases, while the individual books within each class are arranged alphabetically by their authors' names. On the gallery shelves above the modern book room are the works published since 1914, but more than 25 years old. This modern book room is provided with two card-catalogues, describing all the books in the Library published since 1914. The cards in these are arranged alphabetically (1) under authors' names, and (2) under subject headings. This modern catalogue is the first of a series of such sectional catalogues, which it is planned to extend by degrees to the whole Library. The information is equally full on each of the cards concerned with any given book, and the subject-headings conform with those of the *Quarterly Cumulative Index Medicus*. On all cards in both catalogues is indicated the class in which each book should be looked for on the shelves. It is

hoped that readers will thus have a minimum of trouble with cross-references from one card to another, and that uniformity of subject headings with the familiar Index Medicus code will be an added convenience. The author catalogue is complete up to date, but the subject catalogue is still in process of compilation for the books published before and during the war.

At a future date it is intended to classify the books in a more logical sequence of related classes than the present alphabetical arrangement. Such a classification has recently been completed for the arrangement of the books published between 1850 and 1914. These are now shelved round the gallery of the Reading Room according to the lucid classification schedule elaborated by C. C. Barnard,[2] which has proved itself in several libraries more easy to use than the medical schemes used in general libraries. Under Barnard's scheme the older books are segregated into historical period classes, while the classes of modern books are logically arranged, beginning with the sciences and progressing through the general aspects of medicine to the special literature of individual systems and organs of the body. Until 1938 the books in the College Library were almost wholly unarranged, and were placed on the shelves by occasion of their acquisition, the newest arrival, whatever its date or subject, being placed in the first vacant space. Books could only be found by reference to the catalogue which recorded the shelf-mark or fixed place in which the book stood. Thus a book of the sixteenth century might be housed beside a book published last year, while two modern books on the same subject might be separated by many of totally unrelated matter.

The books have now been sorted into period groups, following the Barnard schedules. As was stated above, those published after 1850 have been classified, but the earlier books are being arranged within each period in an alphabetical series by authors' names. This has already been completed for those printed before 1700. In due course each period group will have its two catalogues of authors and subjects. The remaking of the catalogues is, however, a long-term project, only that for the early, pre-1700, books is at present in hand concurrently with the completion of the modern, post-1914, catalogue.

In the meantime all the pre-1914 books, and with them the large collection of pamphlets and also the manuscripts, are included in the old " dictionary " card-catalogue accessible to readers in the main Reading Room. In this the author and subject cards are arranged in one continuous alphabet, but the subject-entries are of the briefest nature and reference has to be made from them to the author-entries, which are themselves quite brief, in order to trace any particular book.

[1] ROYAL COLLEGE OF SURGEONS (1947) The Library, periodicals and their use. *Ann. Roy. Coll. Surg.*, 1, 273-274.

[2] BARNARD, C. C. (1936) *A classification for medical libraries.* London, Lund Humphries.

DIARY FOR MAY

(17th—31st)

Mon. 17   College closed.

Tues. 18   3.45   DR. G. L. BROWN—Neuro-muscular Transmission.
          5.00   MRS. E. K. DAWSON—Carcinoma of the Breast.

Wed. 19   Final Fellowship Oral Examination (Ophthalmology and Oto-laryngology) begins.
          3.45   DR. G. L. BROWN—Neuro-muscular Transmission.
          5.00   MRS. E. K. DAWSON—Sarcoma of the Breast.

Thur. 20   Final F.R.C.S. Examination (General Surgery) begins.
          3.45   PROF. J. H. GADDUM—Drugs Allied to Adrenaline.
          5.00   DR. CUTHBERT DUKES—Tumours of the Rectum and Colon.

Fri. 21   D.A. Examination begins.
          3.45   DR. CUTHBERT DUKES—The Kidney, Bladder and Prostate.
          5.00   PROF. J. H. GADDUM—Drugs Allied to Choline.

Mon. 24   Final Fellowship Oral Examination begins.
          3.45   PROF. T. NICOL—Selected Features in the Anatomy of the Pelvis and Perineum.
          5.00   DR. J. R. M. INNES—The Comparative Pathology of the Central Nervous System.

Tues. 25   3.45   PROF. F. WOOD JONES—Visceral Outlets of Hind End.
          5.00   MR. F. F. RUNDLE—Arris and Gale Lecture—The Anatomy of Exophthalmos.*

Wed. 26   5.00   PROF. A. C. LENDRUM—The Surgical Significance of some so-called Simple Tumours.

Thur. 27   5.00   PROF. A. C. LENDRUM—The Surgical Significance of some so-called Simple Tumours.

Fri. 28   3.45   DR. D. V. DAVIES—Knee Joint.
          5.00   DR. A. C. CROOKE—Adrenal Cortical Hormones.

Mon. 31   3.45   PROF. F. C. HAPPOLD—Growth Factors.

DIARY FOR JUNE

Tues. 1   4.00   PROF. E. D. ADRIAN—Central Nervous System.

Wed. 2   4.00   PROF. E. D. ADRIAN—Central Nervous System.

Fri. 4   D.I.H. and D.L.O. Examinations (Part I) begin.

Mon. 7   5.00   PROF. IAN AIRD—The General Approach to Children's Surgery.

Tues. 8   5.00   MR. C. DONALD—Surgical Conditions in the Neck in Childhood.

Wed. 9   5.00   MR. H. EDWARDS—Pyloric Stenosis and Hirschsprung's Disease.
          7.00   Monthly Dinner for Fellows, Members and Licentiates.

Thur. 10   D.P.M. (Part I), Pre-Medical and Final L.D.S. (Part I) Examinations begin.
          5.00   MR. T. TWISTINGTON HIGGINS—Surgery of the Upper Urinary Tract.

Fri. 11   D.I.H. and D.L.O. (Part II) Examinations begin.
          5.00   MR. J. M. BROWN—Traumatic Surgery, including Burns and Scalds.

Mon. 14   5.00   MR. E. LLOYD—Some Fractures in Childhood.

Tues. 15   5.00   SIR THOMAS FAIRBANK—Abnormalities of the Skeleton.

Wed. 16   5.00   SIR LANCELOT BARRINGTON-WARD—Acute Abdominal Emergencies.

Thur. 17   First Membership Examination begins.
          5.00   MR. G. H. MACNAB—Surgery of the Newborn.

Fri. 18   D.P.M. (Part II) and Final L.D.S. (Part II) Examinations begin.
          5.00   MR. H. P. WINSBURY WHITE—Surgery of the Lower Urinary Tract.

Mon. 21   5.00   MR. DENIS BROWNE—Hernia and Undescended Testicle.

Tues. 22   5.00   MR. T. H. SELLORS—Chest Surgery.

Thur. 24   5.00   PROF. A. SORSBY—The Dystrophies of the Retina and Choroid—Recessive Affections.*

Fri. 25   D.P.H. Examination (Part I) and L.D.S. Examination (Special Anatomy and Physiology) begin.
          5.00   PROF. A. SORSBY—The Dystrophies of the Retina and Choroid—Dominant and Sex-linked Affections.*

Tues. 29   Final Membership Examination begins.
          5.00   PROF. H. J. SEDDON—Bone Growth.
          6.15   DR. S. ROWBOTHAM—The Relation of Endocrine Imbalance to Anæsthesia.

Wed. 30   5.00   MR. H. O. CLARKE—Strains and Sprains.
          6.15   DR. A. H. GALLEY—Caudal Analgesia.

*Not part of Courses.

THE ASSOCIATION BETWEEN CERTAIN ANATOMICAL FACTS, NORMAL AND MORBID, AND THE SYMPTOMATOLOGY OF INTERVERTEBRAL DISC PROTRUSIONS IN THE LUMBAR REGION

Hunterian Lecture delivered at the Royal College of Surgeons of England

by

Harvey Jackson, F.R.C.S.
Surgeon, National Hospital for Diseases of the Nervous System, Queen Squ

on

10th July, 1947

IT IS NOT the purpose of this lecture to offer a detailed and exhaustive discourse on the symptomatology of Disc Protrusions in the lumbar region, but rather to indicate some of the attendant factors, anatomical and/or pathological, affording explanation of certain clinical features manifest in patients harbouring symptomatic protrusions. It will be noted that the term " symptomatic " is used for these lesions ; it is used advisedly because the investigations of Andrae[1] revealed the existence of such cartilaginous excrescences in some 15.2 per cent. of 365 consecutive post-mortems on unselected material, and the writer from time to time has seen similar conditions at autopsy. This fact bears mention also as a possible negation to the impression held generally as regards the resting of the causation in injury, though this is no immediate part of the present theme.

Review of the Literature

To review the literature on this subject is to be impressed by the delayed appreciation of the association between sciatic pain and the existence of a disc protrusion. It was in 1934 that Mixter and Barr[2] published that admirable and enlightening paper of far-reaching consequence calling attention to the relevancy of symptons and source. But in 1911 Goldthwaite of Boston[3] suggested the possible significance of the prominence of an intervertebral disc as an agent in sciatic pain production. Moreover, this paper contained an illustration of a typical protrusion, as well as a photograph of a patient depicting that obliteration of the lumbar hollow commonly displayed in the presence of this disorder.

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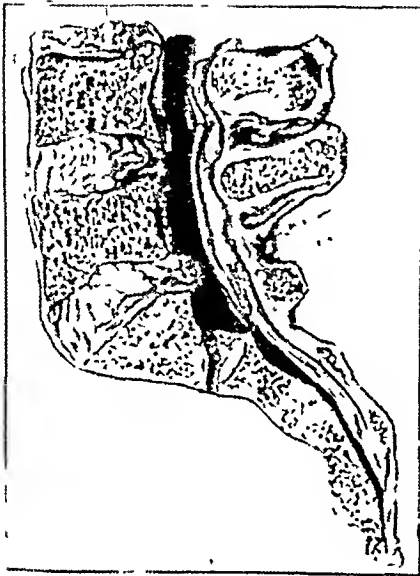
Fig. 1. Illustration from the paper by Goldthwaite showing a diagram of a typical Disc Protrusion and a photograph of the obliteration of the lumbar hollow so commonly associated with this lesion.

Much about the same time Middleton and Teacher of Glasgow[4] described an extrusion of disc tissue within the spinal canal responsible for a paraplegic condition. One cannot help thinking that, had adequate attention been paid to the observations of these authors, many cases of sciatica might have secured more lasting relief, and the conclusions of Mixter and Barr have been anticipated by a number of years. Renewed interest obtruded between 1925 and 1934[5] with the appearance of a number of papers recording cases in which there existed the association of spinal compression, or spinal root disturbance, with cartilaginous masses in the spinal canal. The pathological interpretation, however, favoured a neoplastic origin, and the term " chondroma " was usually applied, although one writer, Von Pechy[6], was not content with this designation, preferring to use the term " ecchondrosis." Cases of this nature were not unknown in this country ; well does the author recollect more than one such operative disclosure during the time that it was his privilege to work with the late Sir Percy Sargent at The National Hospital, Queen Square.

The acceptance too readily of preconceived ideas is perhaps one explanation for misguidance in these matters. One finds difficulty in citing a more apt instance of putting the cart before the horse than is apparent in the term " sciatic scoliosis " with which all of us are so familiar. Here is a term implying that the sciatic element is creative,

whilst the disturbance at the source is inferred as consecutive. Some further comment on this matter will be made later in the lecture.

The Relationship between Sciatica and Disc Protrusions

Before proceeding further it will not be amiss to consider the true relationship between sciatic pain and the incidence of Disc Protrusions. Already one has referred to the existence of protrusions probably of no clinical significance whatsoever. Well may one enquire as to what proportion of cases of sciatica originate in this pathology? Certainly the terms " sciatica " and " disc protrusion " are not reciprocal. It is generally accepted that disc protrusion is the commonest single cause of sciatic pain. Perhaps the investigations of Henderson[7] at the Mayo Clinic bear worthy presentation! This observer enquired into the causative processes in some 10,000 cases of lumbo-sciatic pain, and noted that only 1.73 per cent. were of disc origin.

It is a platitude that accuracy in diagnosis is essential to correct treatment and a satisfactory result. Rarely is this better shown than in the case of sciatica arising out of a disc protrusion. With the object of accurate diagnosis to the fore, in the earlier years some form of verification proved necessary, and this amounted to investigation by contrast radiography. Preparations of iodised oil have been injected intrathecally. The disadvantages of these irritating oily substances soon called for the use of inert media, with the consequence that air and more limpid fluids (Pantopac, Myodil, etc.) have been applied. In this way there evolved a clinical syndrome whereupon a reliable diagnosis is to be formulated and accessory investigations discarded. The basic factors in this syndrome essential to the diagnosis are :—

1. A remittent or intermittent history.
2. Changes in the posture and mobility of the lumbar region of the spine.
3. Sciatic manifestations :
   (a) Pain of root type ;
   (b) Lasegue's Sign.

It should be noted that pain in the back is not an essential factor. Indeed many a patient has flatly denied the existence of back pain at any time.

In order that the level and location of the protrusion may be assessed, other or accessory data are required. These will be given consideration in due course. Plain X-rays, however, are advisable in exclusion of unsuspected bone disease, and in revealing the existence of structural abnormalities of developmental nature.

The Significance of Spinal Manifestations

Recognition of the spine as the seat of the primary lesion, though ignored in earlier years, would appear more or less obvious on our present understanding.

Nowadays it is strange indeed to read of sciatic scoliosis in such an authentic work as Whitman's Orthopædic Surgery[8], and to see in this book an illustration of this deformity showing the typical distortion that attends a disc protrusion. Two important points emerge :—first of all, that the back as the source of the syndrome could have been ignored, secondly, what explanation is forthcoming for the peculiarly diffused nature of the resultant distortion ? The answer to the first problem has been suggested as due to erroneous teaching formulated on preconceived ideas ; with regard to our second problem, one may well enquire as to how a lesion situated lateral to a particular nerve root could possibly excite spasm in a muscle whose segmental nerve supply is that of a more cranial level ? Perhaps the solution is afforded in the recurrent branches of spinal nerves, as described by Spurling and Bradford[9]. These authors have drawn attention to the existence of this special branch of the lumbar spinal roots ; the branch is given off from the root after the root has vacated the spinal canal, it re-enters the canal through the intervertebral foramen, then descends in the extradural tissues to become distributed to the tissues overlying the posterior aspect of the intervertebral disc two vertebral segments in a caudal direction.
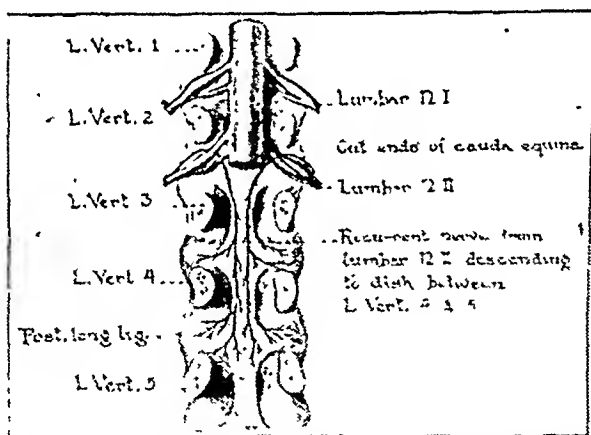
Fig. 1.—Distribution of the second recurrent lumbar nerve to the region of the third and fourth lumbar vertebrae and the fourth and fifth intervertebral disks.

Fig. 2. Diagram from the paper by G. Spurling and F. K. Bradford.

In this way the extradural tissues overlying the fourth lumbar disc gain a nerve supply from the second lumbar root. A disc lesion may thereby initiate irritative phenomena over a region represented by four or even five neural segments. A protrusion at the level say of the fourth lumbar disc, therefore, should be able to account for primary disturbance in the spine up to the second lumbar vertebra, and the loss of the lumbar hollow thereby be rationally explained. Other forms of distortion may

Fig. 3. Extensive changes in posture consequent on a Single Disc Protrusion—so-called "Sciatic Scoliosis."

Fig. 4. Photograph of patient showing complete obliteration of the lumbar hollow.

be derived in like manner, e.g., lateral deviation, scoliosis, and restricted mobility.

This recurrent branch of a spinal nerve is an important discovery, but its presence cannot be detected in the living. We are well aware of the intercommunicating trunks between somatic nerves in the various plexuses, e.g., cervical, brachial, and lumbar, but it is not common knowledge that connecting links are at times to be seen within the spinal meninges. The author has observed such neural links on a number of separate occasions during exploration of the meninges with a view to resection of the appropriate posterior spinal root. These nerves have been seen to run sometimes between consecutive roots, sometimes between alternate roots. The condition is somewhat singular for a symmetrical pattern does not always occur. Perhaps an additional or equivalent factor in the diffusion of signs and symptons is to be found therein.

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Localisation of the Faulty Disc

Change in Sensory Perception.

When approaching a more intimate analysis of the attendant signs one cannot always evince satisfaction as to the existence of sensory depression, or suppression, in a given case. Some observers explain a lack of sensory change on the involvement of but one nerve root. Whether or not this can be so remains uncertain, but if it be so, the writer finds difficulty in explaining certain phenomena about to be described. When diminution in sensory perception is objectively recognisable, the proximal border of the affected zone bears importa as an accurate indicator of the level of the causative lesion. Thus a border of alteration where fourth and fifth lumbar dermatomes unite designates the fifth lumbar root as the level concerned. This root accepts intimate relationship with the fourth lumbar disc for at this level the root migrates from the rest of the cauda equina ; it accepts no apposite relationship with the fifth lumbar disc. We are all conversant with the usual diagrammatic representation of the cutaneous distribution of the somatic nerves ; but in my submission, no single picture is acceptable as absolute. That different distributions are indicated as arising from any one root at first sight would seem unwarranted ; yet regular registration of areas of dysæsthesia arising out of pathological states of a root, or from operative removal, reveals variable clinical pictures in different individuals. In fact, it would appear that involvement of the fifth lumbar root can bring about one of three different sensory patterns.
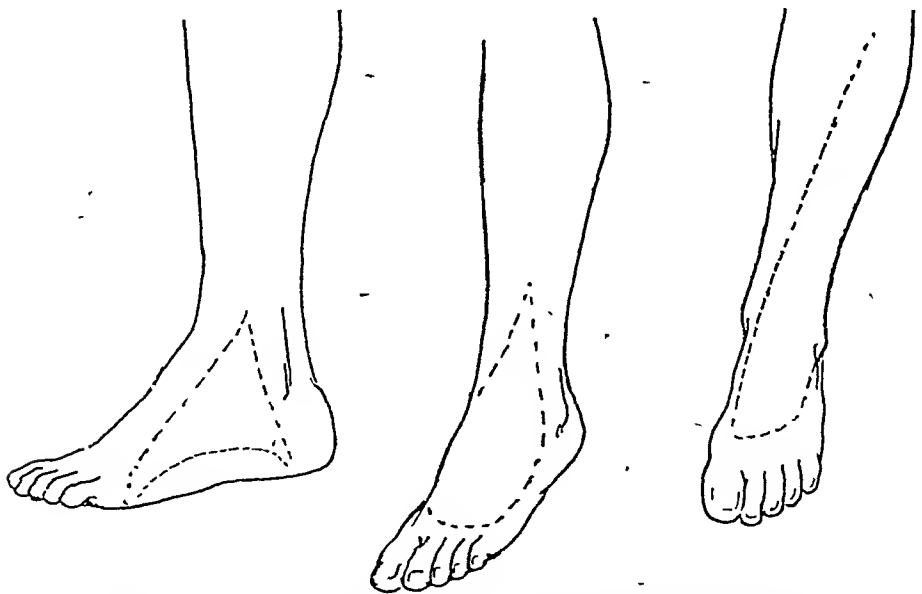
Fig. 5. Diagrammatic representation of the three types of sensory area associated with the fifth lumbar nerve in different patients.

How are we to reconcile this view on anatomical grounds? While prefixed and post-fixed origins are generally recognized in so far as the brachial plexus is concerned, we are less appreciative of a comparable variability of the lumbo-sacral plexus. Again, skeletal variations in the forms of sacralization of the fifth lumbar vertebra and lumbarization of the first piece of the sacrum are commonly encountered. It is the writer's contention that herein lies the factual explanation of the several patterns of possible sensory disturbance. One may go further, perhaps, in interpreting a particular sensory chart as indicative of one of these structural formations of the spinal column in the person concerned. In other words, existence of sacralization or lumbarization may be indicated by the form of the attendant zone of sensory disturbance consequent on implication of the fifth lumbar root. There are occasions when one remains in doubt as to the pattern of sensory imperception, so much so that differentiation of the fifth lumbar root disturbance from that of the first sacral root would appear to be uncertain. When confronted by this, recognition of the first sacral and fourth lumbar border over the medial margin of the foot may verify the actual level.

Disturbance of Motor Power and Tone

Motor disturbances indicate the spinal level at fault. According to whether the disturbance of function involves the anterior or the posterior compartment of the leg, the fifth lumbar root or first sacral is indicated respectively. Although wasting and flabbiness of muscles are almost constant features, gross weakness is uncommon. Usually only extension of the great toe or other digits is involved.

Reduced or Suppressed Reflex Activity

When interference with reflex function is under consideration, although the knee jerk is occasionally depressed, it is the ankle jerk that claims our main attention. Diminution of the knee jerk is an interesting phenomenon, not easily explainable, but this may possibly arise from pre-fixation of the spinal roots. It is to be seen in association with a protrusion at the fourth lumbar disc. The reduction or suppression of the ankle jerk implies dysfunction in the first sacral root. Although this is more likely to occur from a lesion at the level of the fifth lumbar disc, it may result from a lesion at the fourth disc. Hence, the spinal level at fault must be assessed on coincident signs. No matter what the associated signs may be, the level of the lesion is that of the most cranial root involved.

Localization in respect of Nerve Roots

At this juncture it will be appreciated that the level of a disc protrusion should be discernable from these various sensory, motor, and reflex indications. But a more comprehensive understanding of the distribution of disc protrusions is essential to appropriate treatment. In respect of

any one disc there are five common sites : one is central, the others are constituted in two comparable sites on the left and right hand sides. The two possible lateral sites rest lateral or medial to the root originating from the cauda equina at that particular disc. For purposes of simplification these are described as para-radicular or para-caudal protrusions.

A. *Para-radicular*   B. *Para-caudal*   C. *Central*
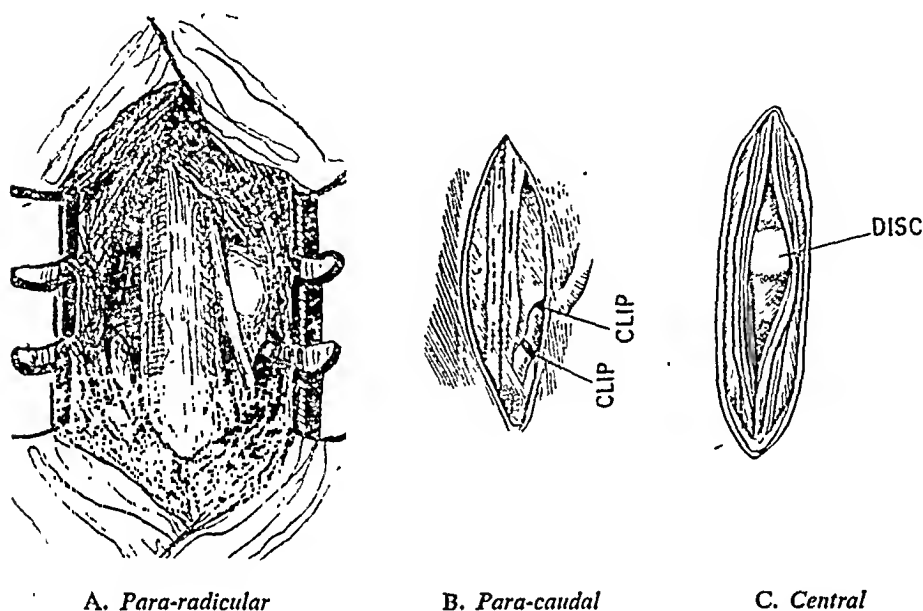
Fig. 6.   Diagrams showing the situations of protrusions as described in the text.

A refinement in diagnosis is the recognition of the precise situation of any protrusion.   The central type is surmised on the existence of bilateral symptoms or signs. A para-radicular protrusion is indicated by a limitation of sciatic manifestations to one root, i.e., the fifth lumbar root in the case of the disc between the fourth and fifth lumbar vertebræ, the first sacral root if the disc between the fifth lumbar vertebra and the first piece of the sacrum is responsible. Verification of this is sought in the effect of forward and lateral bending, as accentuation of pain usually accompanies deviation to the side of the sciatic pain. On the other hand, a para-caudal protrusion shows interference with more than one root and the pain tends to be aggravated by lateral bending away from the side of the pain. In other words, a para-radicular protrusion at the level of the fourth lumbar disc is evidenced by fifth root signs, no depression of the ankle jerk, and increased discomfort when bending to that side. A para-caudal protrusion should incite fifth root signs, evidence of first sacral root involvement, and, perhaps, other sacral roots, together with additional discomfort when bending towards the opposite side.   It is

possible for a large para-caudal protrusion to produce a bilateral distur-
bance, but the intensity of the symptoms is not equal in the two lower
limbs.

Fig. 7. Photograph of a para-radicular
protrusion taken at operation.

The direction of the protrusion in respect of the lateral types would
appear to be controlled by the lateral process of the posterior longitudinal
ligament. The para-radicular swelling occurs above this lateral process,
whereas the para-caudal type extends below it. As the posterior common
ligament affords additional support for the medial protrusion, it is usual
for the mound so formed to be less pronounced than is the case with either
of the lateral protrusions.

Multiple Protrusions and Their Significance

Multiple protrusions have been recorded by many surgeons, indeed
the late Dr. Walter Dandy[10] described them as present in 20 per cent.
of all disc cases. Another matter of interest is the incidence of protrusions
at the common levels. Some surgeons state that the fourth lumbar disc
is the commoner level whereas others have found the fifth disc a more
common source. For a satisfactory operative result it is essential that the
level of the clinical disorder be selected; indeed, unless the clinical state
coincides with the operative disclosure, dissatisfaction may result. As
we have already noted, disc protrusions of no clinical significance are to
be found in quite a high percentage of individuals. Should one of these
be found at operation and accepted as responsible in place of the actual
cause, an unsatisfactory result is to be expected.

Pathological Changes in the Affected Nerve Roots

Remittency or intermittency of symptoms is not only a common
occurrence in a case of Disc Protrusion, it is an essential factor in the diag-
nosis. How this alternation arises is not really known, but one assumes
either a variability in the dimensions of the protrusion, or some associated

changes within the affected nerve or nerves. Trauma is a common precipitant of an attack, but acute fevers (e.g., influenza, tonsillitis) and pregnancy undoubtedly play their part[11]. But it is not to the protrusion alone that we must pay attention. Changes within the nerves are also to be seen. On several occasions one has been able to demonstrate hæmorrhage into the nerve subsequent to a severe and recent attack. It may be said that such a finding is merely a result of surgical manipulation at the time of operation. That this could be the case would probably require such hæmorrhage to be much more frequently present in resected roots; moreover, the histological sections can be seen to contain fibrinous exudate and pigmentation, both products of the effusion of blood, and these require a period of time to develop; therefore, operative trauma cannot be the reason for all such changes (See Fig. 8.)

Evidence of chronic degenerative processes also occurs in the nerves; demyelination, fragmentation of the axons, vacuolation and complete disintegration. These changes are not usually found to involve the whole posterior root, but only certain fasciculi. (See Fig. 9.)

In this there is another possible explanation for the alternation of the history, for fresh fasciculi could be affected on each occasion and not until the whole root is completely degenerated would a permanent and natural cure result. It is for this latter reason that the author has resected the posterior root in a large percentage of cases of positive disc protrusion. Not until the whole posterior root has been excluded is it possible for one to reassure a patient that further sciatic pain is impossible in respect of that particular lesion. (See Figs. 10 and 11.)

Hypertrophy of the Ligamentum Flavum

Gross thickening in the ligamentum flavus is a regular accompaniment of a symptomatic protrusion. Moreover should one consider it advisable to explore more than one intervertebral disc, it is usually at the level of the thickest ligamentum flavum that the protrusion is to be encountered. The part played by this ligament is uncertain, it has even been looked upon as the exciting factor in a patient's symptoms, when other adequate explanation is found wanting. Under such circumstances moral justification for the operation has its part to play in the surgeon's acceptance of this as a possible cause. In the writer's own humble opinion, the ligamentum flavum is most unlikely to contact any spinal root unless that root is distorted from its regular path. What then are the significance and explanation of this hypertrophic change? (See Fig. 12, facing page 283). With the distortions in the back to which we have already referred, an obvious tension must be developed in this ligament. Our daily activities of necessity put intermittent stress and strain on this structure and, in the presence of distortion of the spinal axis, undoubtedly such physical forces must be exaggerated. Increasing and intermittent stress would account admirably for this hypertrophy.
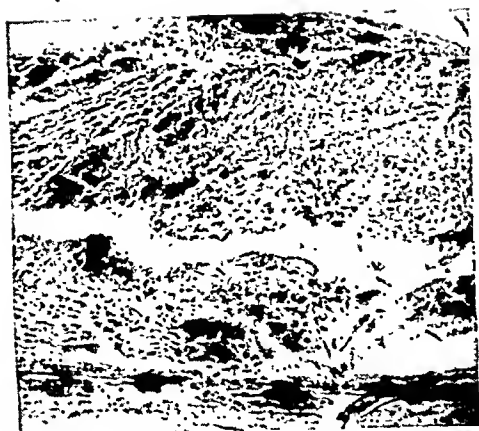
Fig. 8

Section with patches of necrosis and fibrinous deposit somewhat dispersed throughout the nerve. × 64.
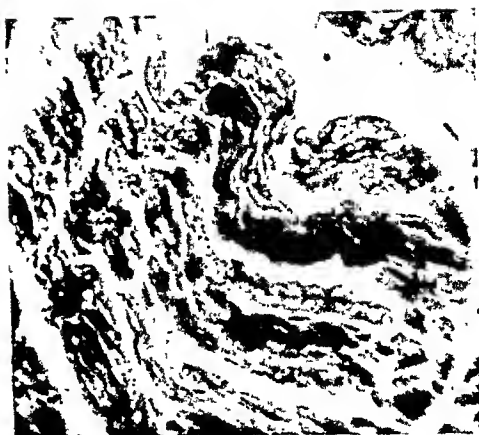
Fig. 9

Section of nerve showing demyelination with vacuolation of myelin sheath. × 250.
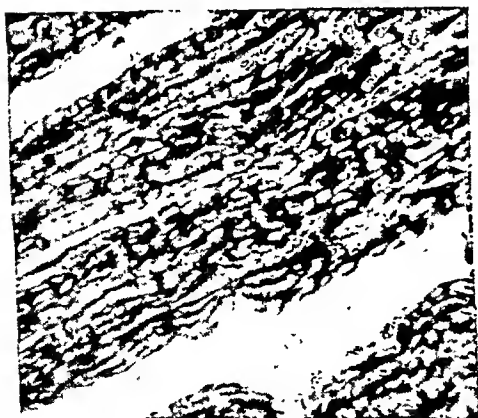
Fig. 10

Section showing very extensive demyelination. × 250.
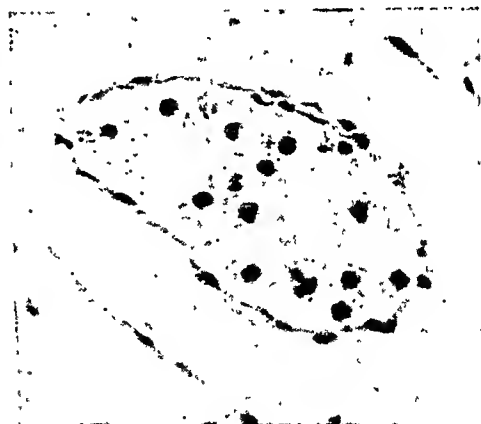
Fig. 11

Section of a capillary with some hæmorrhage between the endothelial lining and surrounding areolar tissue. Very fine pigment can be seen in the endothelial cells and also in neutrophil polymorphs. This indicates old hæmorrhage in the vicinity. × 500.

× 5
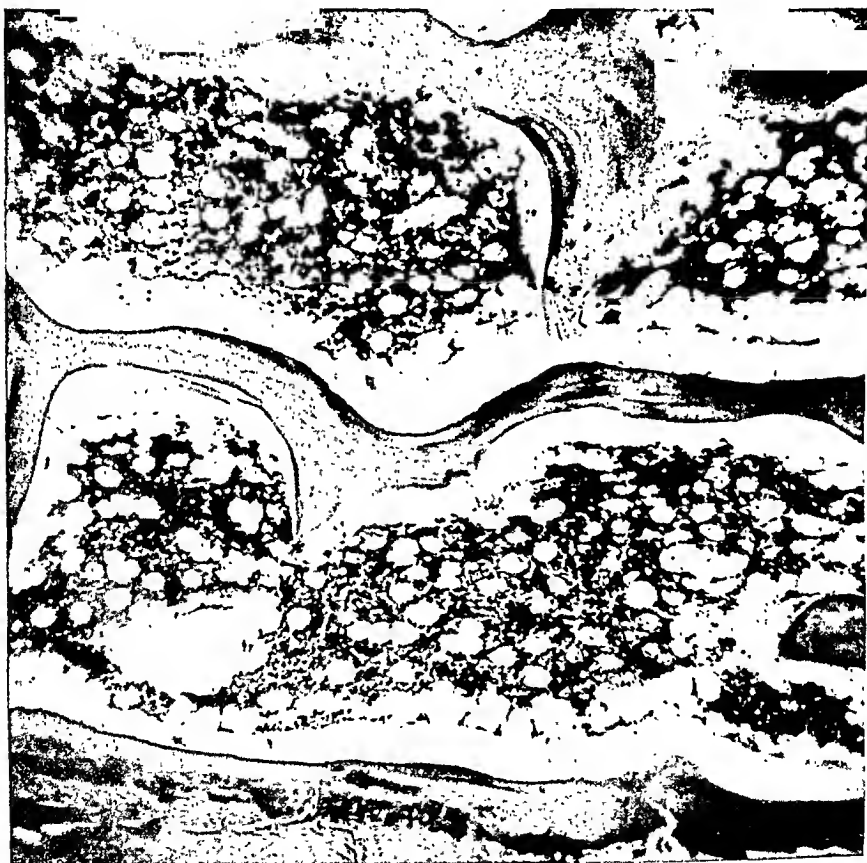
× 64

Fig. 12

Sections of a " protrusion " consisting of encapsulated cancellous bone.
A specimen not likely to result from trauma.
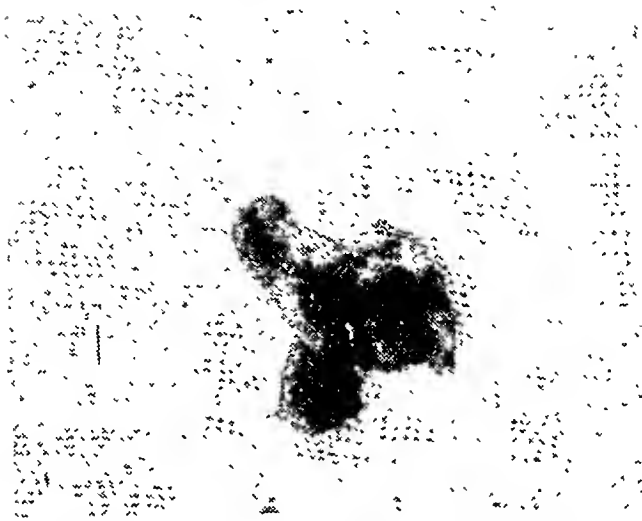
Fig. 13. A much hypertrophied Ligamentum Subflavum excised from the level of the Disc Protusion.
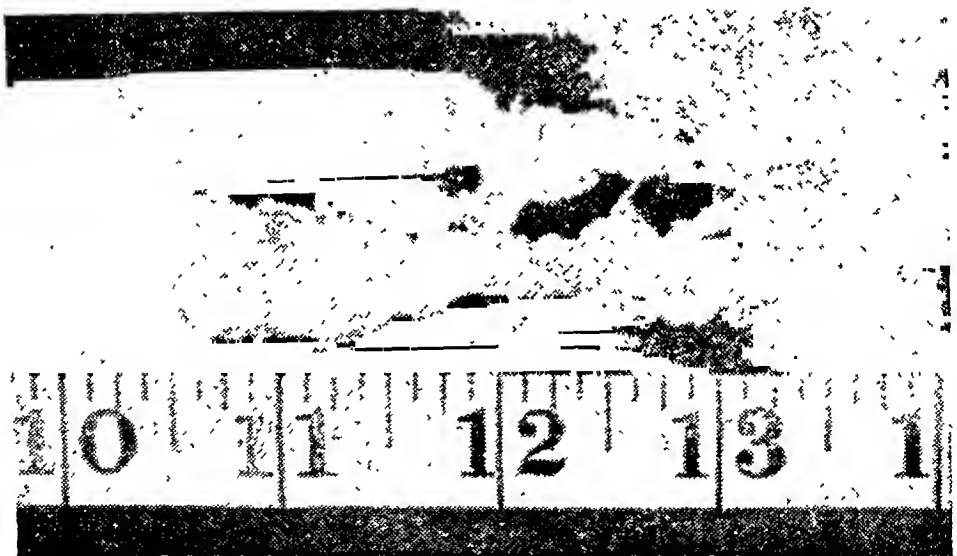
Fig. 14. An unusually large portion of disc tissue found completely sequestrated at operation.

Destructive criticism would discourage progress but a picture veiled by misconception may lead one away from rational interpretation. So many terms are applied to these protrusions : retropulsion, rupture, herniation, prolapse, each inferring some different mechanism. On our present-day knowledge no one of these processes is a proven quantity. Surely these terms are misleading, and it is for that reason that the writer has applied the term Disc Protrusion throughout. In fact, it is the writer's opinion that these defects most likely are of developmental nature, possibly

increased by other levers in the forms of trauma, toxæmia and pregnancy. However, evidence in favour of this hypothesis is not within the scope of this communication.

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THE ANATOMY OF INGUINAL HERNIA

The Substance of an Arris and Gale Lecture

Delivered at The Royal College of Surgeons of England

by

H. F. Lunn, B.Sc., M.B., F.R.C.S.

Formerly Sir Halley Stewart Research Fellow, Royal College of Surgeons of England

" *No disease of the human body belonging to the province of the surgeon requires in its treatment a greater combination of accurate anatomical knowledge with surgical skill than Hernia in all its varieties.*" (Astley Cooper, 1827.)

THERE NEED BE no lack to-day of surgical skill. The increased scope of surgical practice and the development of anæsthesia have provided every surgeon with the opportunity to attain a high standard of technical ability. Pressure of clinical work has, however, prevented a parallel advance along the other path which Astley Cooper regarded as essential—that of accurate anatomical knowledge.

We must admit that there has been no significant advance in the understanding of the structure of the human inguinal region during the past century. The operations of Bassini (1890), Gallie (1924) and others are all based upon the traditional conception of the anatomy of this region, handed down from the time of Falloppius (1561). Views held by many centuries of experienced observers cannot be lightly dismissed, but we do well to retain them only if they survive repeated examination in the light of modern knowledge.

Perhaps in no other region are conflicting views more strongly held and contested. To one surgeon the transversalis fascia is all-important, to another it does not exist as a constant layer. Some believe that the cremaster is redundant, others that it is an essential part of the inguinal mechanism. In some clinics the patients walk on the day following operation, in others they remain in bed three weeks. One surgeon attends closely to the superficial inguinal ring and the external oblique aponeurosis, another concentrates on the deep ring. In the same issue of the *British Medical Journal* (August 11th, 1945) is an article deploring the relapse rate after the Bassini operation, and another claiming that this operation gives consistently the best results.

In the midst of such confusion there has been evidence in recent years of an attack against the complacency which has been hampering progress. Sir Heneage Ogilvie (1937) has repeatedly stimulated original thought by reasoned criticism of traditional ideas. Harold Edwards (1943) has

abstracted and reviewed the mass of literature concerning inguinal hernia and its repair, and has steered a middle course through the extreme views given above. Lytle (1945) has made a useful contribution by laying emphasis on the internal abdominal ring. His description of the transversalis fascia does not, however, agree with the findings of Douglas (1730) and has not been widely accepted. Anson and McVay (1937, 1940) have presented a conception of the anatomy of the groin which conflicts in several respects with current descriptions, yet which is in accord with the results of the comparative approach.

The inguinal canal has been studied in animals by John Hunter (1786) and by Berry Hart (1910) in relation to the descent of the testicle. Sir Arthur Keith (1923) considered the inguinal canal from a comparative point of view in relation to hernia, but he and subsequent workers (e.g., Miller, 1947) tended to restrict their studies to a comparison between man and the other primates. There is need therefore of a study of the inguinal canal in a wide series of animals, with the application of the conclusions to the problem of hernia. The following pages record the findings that have resulted from an attempt to meet this need through the approach John Hunter would have used.

The Terminology of Hernia

The changing language in which we have to think, to learn, and to teach, is a feature of most of the arts and sciences to-day. Structures acquire an undeserved importance when tradition honours them with a high-sounding name. There is an attack to-day upon eponyms both in anatomy and surgery. The student and the clinician alike resist dictatorship and have chosen to retain certain well-tried eponymous terms. Ligamentum inguinale is often the inguinal ligament, but more often Poupart's. Gimbernat's ligament may also be called the lacunar ligament, but rarely the more recent " pectineal part of the inguinal ligament." Thus the confusion continues.

A time of change is also a time for discrimination, for retaining the useful and for discarding what has ceased to be effective. Should all eponymous terms be discarded ? If the alternative is a heavily sounding Latin word or a discursive phrase, some names should indeed be spared. We are in danger of forgetting the great pioneers in anatomy and surgery when we extract all their names from our books and teaching.

Hesselbach (1806) stressed the inferior epigastric artery as a landmark, but the remainder of his triangle serves no further purpose. His ligament (interfoveolar ligament, Gray, 1946) is described as a thickening in the transversalis fascia, but is best considered, together with that of Henle, as a part of the conjoined tendon (Douglas, 1890).

It will not be easy to separate Gimbernat from his ligament in clinical teaching. The " pectineal part of the inguinal ligament " is too long a

phrase to survive in these days of speed and brevity of expression. The alternative to the eponym is the lacunar ligament (B.N.A.), a pleasant descriptive term that still appears in Gray (1946). Colles, on the other hand, is immortalised by his fascia and does not need his reflected inguinal ligament, a structure that is found by Anson and McVay (1937) to be a rarity. The name of Nuck (1691) will probably remain attached to the processus vaginalis of the female.

Few would contend the use of Sir Astley Cooper's name for his ligament of the pubis. Sir Astley did not in actual fact give the first description of this ligament, as Gimbernat (1793) had anticipated him. It was Sir Astley Cooper, however, who applied his ligament to the surgery of this area and who thus merits a place among those whose names are retained in our nomenclature. The pectineal line of the pubis is overburdened with the attachment of the pectineus, the pectineal fascia and the pectineal part of the inguinal ligament. The addition of a pectineal ligament (B.N.A. revised) will surely increase the confusion. The ligament of Cooper, if retained, would draw attention to these fibres of importance to the surgeon and to the memory of the one who contributed more to the understanding of this region than all the generations which have followed.

There have also been changes in the non-eponymous terms in current use. The entry to and exit from the inguinal canal are no longer abdominal rings. Now they are inguinal rings, deep and superficial (B.N.A. revised), although the older terms, abdominal and subcutaneous inguinal rings (B.N.A.) are also employed. The tendons of the internal oblique and the transversus abdominis become fused near their attachment to the pubis and are now termed the conjoint tendon. This is better than the falx inguinalis aponeurotica of the B.N.A., but I must express a grammatical preference for the old conjoined tendon.

The classification of inguinal herniæ is far from satisfactory. Maclise (1853) and Sir Heneage Ogilvie (1937) have drawn attention to the contradictions created by the terms direct and indirect. An indirect or oblique hernia may stretch the medial edge of the deep inguinal ring so that it emerges almost directly behind the superficial ring. The funicular type of direct hernia may enter the inguinal canal via a defect in the conjoined tendon and pass obliquely along the cord towards the scrotum (Ogilvie, 1937). A congenital or infantile type of hernia may first appear in adult life and the former kind may be acquired.

Should such confusing terms be retained for longer than the hundred years which have already passed since Maclise suggested relating herniæ to the inferior epigastric artery? This artery is accepted as the surgeon's landmark in the diagnosis of hernia. The neck of the sac is either lateral or medial to this vessel. Herniæ could therefore be classified as congenital or acquired medial herniæ, and congenital or acquired lateral herniæ, depending on opinion as to their ætiology.

A Note on the Ætiology of Hernia

" *The elastic tissue forms membranes in the walls of hollow organs upon which a changing pressure acts from within, as in the largest arteries, in some parts of the heart, in the trachea and bronchi.*" (Maximov and Bloom, 1942.)

The abdomen can be included amongst the hollow organs upon which a changing pressure acts from within. By experiments on skin and arteries, John Hunter demonstrated the presence of elastic tissue before histology and chemistry had revealed the nature of the substances concerned. If such tissue is present in the skin and subcutaneous layers of the abdomen, we would expect to find it also in the fascial and muscular layers beneath. The abdomen as a whole enlarges in ascites and pregnancy, the enlargement including a stretching of the rectus sheath and linea alba. Yet after tapping of the ascites and after delivery, these structures return to their normal length if not overstretched.

Thus it would not be surprising to find that the abdomen, subject to changing pressures as is an artery, had a similar structure in its walls. Routine histological examination reveals that a deep as well as a superficial elastic layer supports the anterior abdominal wall, particularly in the region of the conjoined tendon. I am not yet in a position to report the amount of elastic tissue and collagen found in the conjoined tendons of subjects of different age and sex. There is an opportunity for someone to study the composition and congenital variations of the abdominal wall as thoroughly as the arteries have recently been studied in their relation to congenital aneurysm of the cerebral vessels.

Sir Astley Cooper (1830) studied the conjoined tendon after removal of the lower abdominal muscles from fresh post-mortem subjects. In one such specimen he comments on the unusually high proportion of muscle fibres found in the conjoined tendon. Similar blocks of tissue have been taken from human subjects, including the femoral vessels, and examined for the presence of muscle fibres in the conjoined tendon. A variable number of muscle fibres were found, but the proportion of elastic tissue was constantly and unexpectedly high.

The existence of elastic tissue in the abdominal wall and conjoined tendon may assist in an explanation of the facts observed in the onset of hernia. Hernia due to a preformed sac, the patent processus vaginalis of Hamilton Russell (1922), would be expected to appear in infancy or childhood. Approximately 25 per cent. of all inguinal herniæ arise before the age of 15 years. The other congenital defect is a weakness, or perhaps a deficiency in the elastic tissue in the conjoined tendon. This factor, together with trauma, may account for the herniæ which occur between the ages of 16 and 35, and which form 39 per cent. of the total. The two acquired factors, trauma and senile changes in all tissues of the inguinal sphincter, would account for the herniæ arising in middle and old age. In

this scheme I have not differentiated between medial and lateral herniæ, but have quoted the proportions of over 17,000 inguinal herniæ given by Macready (1893).

I do not believe that the part played by trauma will be fully understood until the " ruptures " which arise suddenly during strain are treated as acute injuries and operated upon within a few hours of their appearance. The part played by elastic tissue and its defects and changes will only be clear after the survey of which I have spoken has been completed.

Nature and Surgical Handicraft

Surgical skill and anatomical knowledge were Sir Astley Cooper's criteria of success in operations for hernia. Results are to-day in direct proportion to the quality and technique of the operator. Amongst the numerous methods employed it is impossible to find one that is recognised to give consistently better results than any other. Many of these earlier methods are constantly being revived with modifications, sometimes without reference to the originator. I propose to compare them with the way in which nature overcomes the problem of containing intra-abdominal pressure.

Bassini's operation (1890). This operation and its modifications have no counterpart in nature. The conjoined tendon is invariably attached to the pectineal surface of the pubic bone. In no instance is there any fusion between this tendon and the aponeurosis of the external oblique above the line where these two structures are attached to bone. As the direction of the two sets of fibres differs proximal to their insertion, it may be inferred that there is normally a plane of movement between them. In man there is an increasing weight of evidence to show that a single line of sutures drawing the conjoined tendon down to the inguinal ligament does not result in a persistence of this distortion of anatomy (Gallie, 1924 ; Cowell, 1927). Although we are thus critical of the Bassini type of repair, we must admit that most of our reconstructive operations are based, like that of Bassini, upon the inguinal ligament.

Approximation of the rectus abdominis to the deep inguinal ring. Whether this is achieved by lateral transposition of the rectus abdominis (Blood-good, 1898), or by medial displacement of the deep inguinal ring (Fowler, 1897), the relationships are similar to those of the mammalian quadruped. The weakness of the former group of operations lies in the use of the mobile medial end of the inguinal ligament as the new attachment for the rectus or a flap of its sheath. Transposition into the medial end of the pectineal line of the pubis might prove more effective in restoring normal anatomy.

In Fowler's operation, the posterior wall of the inguinal canal, including the inferior epigastric artery, is incised and the spermatic cord is displaced medially. If the crura of the superficial ring are also displaced medially, the length and obliquity of the inguinal canal will be in part restored.

Whichever of these two methods is used, the result is to approximate the deep inguinal ring to the lateral edge of the rectus abdominis, which thus becomes the posterior wall of the inguinal canal. This advantage is diminished by the fact that the spermatic cord has been placed between aponeuroses instead of the actively contracting inguinal sphincter through which it normally passes.

Lateral displacement of the deep inguinal ring. Schmieden and later Brandon (1945) have described methods of transferring the spermatic cord laterally so that it emerges between the lower fibres of the internal oblique. From this new deep inguinal ring the length and obliquity of the inguinal canal is restored. From the peritoneum to this point of emergence through the internal oblique, however, there is a direct channel into which a new sac may protrude. The sharp alteration in direction of the spermatic vessels may endanger the blood supply to the testis. The natural course of the spermatic vessels is along a surprisingly gentle curve (Denis Browne, 1933).

Replacement of the natural sphincter mechanism. The balance of modern opinion in this country, led by Sir Heneage Ogilvie, is to make full use of the natural sphincter mechanism and to give it every assistance as long as it is deemed competent and is not stretched beyond repair. Should it have failed, it must be replaced by an unyielding barrier of foreign or autogenous tissue. Nature must still provide the framework upon which the barrier can be built and comparative anatomy may assist in the search for firm foundations.

The Comparative Anatomy of the Groin

" *I never see a young lad climbing a steep ladder with a heavy sack of corn on his back without feeling that, but for the grace of a strong and perfectly competent groin, he must quickly become the subject of hernia.*"

In these words Sir Arthur Keith (1924) recognises the perfect competence of the mechanism which defends the groin in the great majority of mankind in spite of the erect posture and the demand of civilization. Current anatomical descriptions on the other hand emphasise the apparent weakness of the human groin (e.g., Gray, 1946).

Although the inguinal region of man is modified from the generalised mammalian type, the same basic principles have been found to underlie the anatomy of all animals with hind limbs (Fig. 1). An inguinal ligament has been found to exist in quadrupeds but this structure in no case forms the foundation on which the security of the inguinal musculature depends (Figs. 2 and 3). The inguinal sphincter mechanism is based upon the bone of the pelvic girdle, a fact that will be emphasised by a study of the mammalian inguinal canal.

The inguinal canal with a patent processus vaginalis. In those animals with a widely patent processus vaginalis and a short inguinal canal (Fig. 4)

much muscle surrounds the canal. The lateral border of the rectus muscle supports the medial wall of the canal because the spermatic cord is directed almost vertically downwards. The internal oblique passes usually behind but sometimes in front of the rectus muscle in the different forms and is often muscular close to the midline. The external oblique is strongly muscular around the emergence of the cord and the crura of the external ring are therefore composed of muscle rather than tendinous fibres. Strong cremasteric muscular loops sweep down from the transversus abdominis muscle. Along such an inguinal canal the testes of the rodent are able to pass in and out of the scrotum during or after the rutting season.

Whether the testis is within the abdomen or in the scrotum, the muscle fibres around the canal contract together with the remainder of the parietal muscles and retain the viscera within the abdomen.

The inguinal canal with a closed processus vaginalis. Aponeurosis rather than muscle surrounds the spermatic cord when the processus vaginalis is closed or almost closed proximally, as in the kangeroos, carnivores and apes. The crura of the external abdominal ring are tendinous and the internal oblique and transversus become thin and tendinous as they approach their attachment to the pubis and rectus sheath. The cremaster continues to be well developed and is derived from both the internal oblique and transversus abdominis.
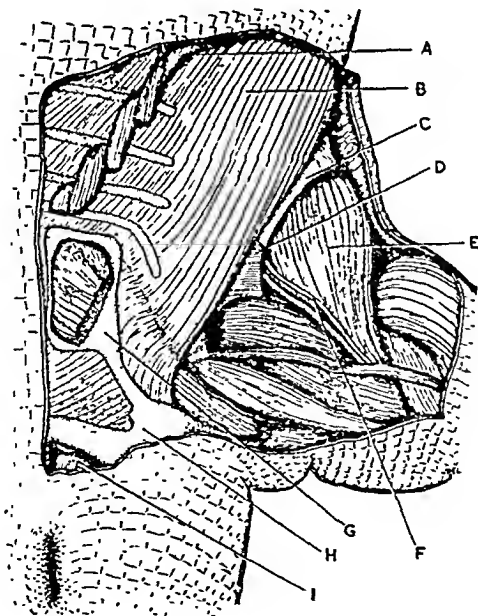
Fig. 1. The groin of a Crocodile (Crocodylus palustris), showing the basic anatomy of the hilum of the hind limb. A. Rectus abdominis, cut through its proximal fibres. B. External oblique, retracted medially. C. Internal oblique attached to a tendinous arch over psoas. D. Transversus abdominis. E. Psoas muscle. F. Femoral nerve (and artery). G. Epipubic cartilage. H. Pubic bone. I. Distal attachment of rectus abdominis.
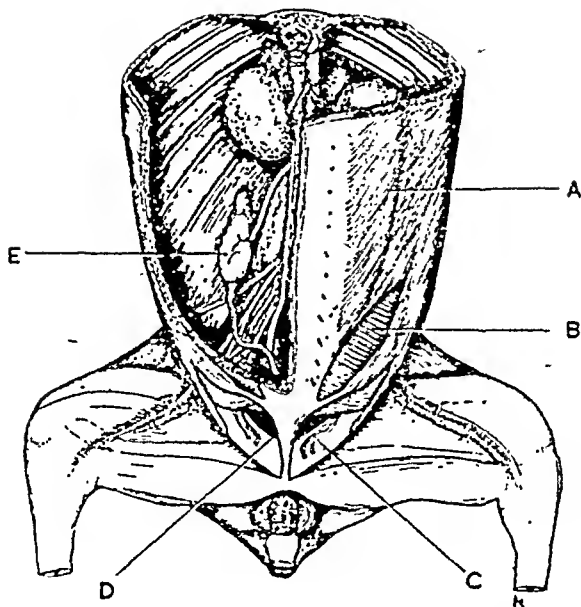
Fig. 2. Dissection of a Hyrax (Procavia habessinica), showing the presence of an inguinal ligament. A. External oblique. B. Internal oblique seen between two of the digitations of external oblique. C. Vessels and a nerve passing into the skin of the groin. D. The thickened, inturned edge of the aponeurosis of the external oblique. E. The testis and epididymis which are permanently intra-abdominal.
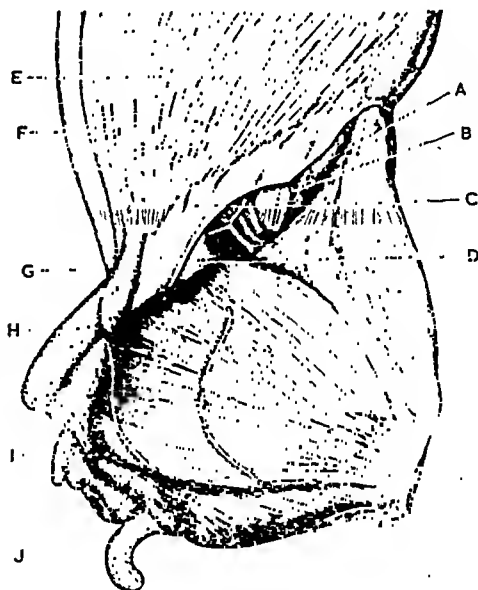
Fig. 3. Lateral view of the groin of a Patagonian Cavy (Dolichotis magellanica). A. Ilio-psoas fascia. B. Psoas major. C. Aponeurosis of the external oblique. D. Pectineal part of the inguinal ligament. E. External oblique, muscular part. F. External oblique, central tendinous part. G. Muscular fibres forming the superficial inguinal ring. H. Testis. I. Penis. J. Tail.
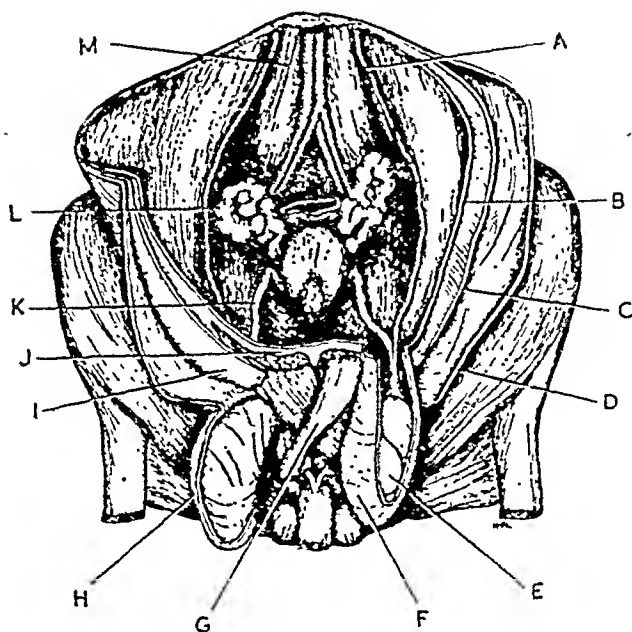
Fig. 4. Dissection of the abdominal wall of a Coypu (Myocastor coypus), showing muscular support to each scrotal pouch. A. Spermatic vessels. B.-J. Peritoneum and transversus abdominis. C.-I. Internal oblique. D. External Oblique. E. Testis. F. Cremaster. G. Rectus abdominis. H. External spermatic fascia. K. Vas deferens. L. Vesicula seminalis. M. Psoas major.

Intermediate types of inguinal canal. Many of the quadrupeds examined are not covered by either of these descriptions which represent the two extremes found. All possible variations are seen. The processus vaginalis usually communicates with the general peritoneal cavity through a constricted neck which may be guarded by a crescentic fold of peritoneum (Giraffe), a fatty pad (Monkey), or a few muscle fibres (Ox).

Comparison With Man

(a) Length and inclination of the inguinal canal. The length of the inguinal canal, when considered in relation to the length of the inguinal ligament, is variable in the present series. In this respect man does not differ significantly from the other mammals. In all forms there is a constant relationship between the degree of patency of the neck of the processus vaginalis, the length of the canal and the amount of muscular tissue in its walls.

In the pronograde quadruped the angle subtended by the two inguinal ligaments has been described as acute. Hence the area of the anterior abdominal wall between the inguinal ligament and the lateral border of the rectus abdominis is relatively small in comparison with the wider angle and larger area in man. This area is guarded by the lower fibres of the internal oblique, transversus abdominis and conjoined tendon and is crossed obliquely by the spermatic cord.

(b) *Composition and attachments of the conjoined tendon.* Reference has previously been made to the interruption in bony attachment of the internal oblique and transversus abdominis to the pelvis below the anterior superior iliac spine. The iliopsoas and femoral vessels are relatively so large in man that the lower abdominal muscles are attached more to the arches over the hilum of the lower limb than they are to bone. Medially these muscles are described as combining to form the conjoined tendon.

The conjoined tendon passes in front of the rectus abdominis and forms the anterior lamella of the rectus sheath. It also passes downwards to reach the pubic crest and the pectineal line of the pubis. Its outer edge is usually so ill-defined that it is seldom possible to indicate the exact line where it terminates (Macready, 1893). The portion of the tendon derived from the transversus abdominis extends further laterally from the pubic tubercle than that from the internal oblique. These two components of the conjoined tendon form the greater part of the posterior wall of the human inguinal canal. The obliquity of the inguinal canal in man is largely due to the obliquity of the insertion of the conjoined tendon into the pectineal line.

Hence, in man the fibres of the internal oblique and transversus below the anterior superior iliac spine have a relatively unstable origin from the wide crural arch and an attenuated insertion into the pectineal line of the pubis which is subject to frequent congenital variations in width and strength.

(c) *Transversalis fascia.* Recent descriptions of the anatomy of the inguinal canal of man emphasize the contribution of the transversalis fascia to the posterior wall and internal abdominal ring. No support for this emphasis has been given by the present study. It has been unusual to find extra-peritoneal areolar tissue or fat separating the peritoneum from the transversus muscle in this area. The peritoneum is usually firmly adherent to the transversus abdominis and to the vas and spermatic vessels as they enter the inguinal canal. No transversalis fascia has been found to exist in quadrupeds as a constant factor in the structure and support of the inguinal canal. In man any strength or resistance in the transversalis fascia is admitted to be due to tendinous fibres derived from the overlying transversus abdominis.

Conclusions

The dissection of a wide series of mammals strongly supports the contention of Keith (1923, &c.) that the inguinal canal is guarded principally by a neuromuscular reflex rather than by a fascial or aponeurotic barrier.

The importance of the abdominal rings has been allowed to exceed that of the sphincteric mechanism in some descriptions of this region.

The internal ring is unable alone or with the support of the transversalis fascia to resist a high intra-abdominal pressure. The external ring is

unable to prevent the emergence of a hernia that has once entered the canal. These rings are guides for the surgeon and landmarks for the dissector, not barriers against internal pressures.

Observations on the Exposure and Repair of the Inguinal Canal

The contribution of Bassini to the surgery of hernia was his exposure of the spermatic cord throughout its length. Celsus, and those who followed him, in attempting to ligate or excise hernial sacs operated from outside the superficial inguinal ring. Due to the lack of anæsthesia and of control of infection, the Middle Ages were rightly called the Dark Ages and surgeons were deterred from operating except for strangulation. Prior to Bassini, subcutaneous operations were coming into favour, having been introduced into England by Spencer Wells and Wood (1863). These were blind manœuvres with attendant dangers and frequent relapses.

Bassini divided the aponeurosis of the external oblique laterally almost as far as the anterior superior spine of the ilium. Medially he divided the superficial ring. The revolt against Bassini is not against his exposure, but against his stitches. I would say that we should carry Bassini's exposure even more deeply if we are to find a firm unyielding basis for our repair.

Beneath the aponeurosis of the external oblique the cremaster forms a thin muscular layer over the spermatic cord. These muscular loops assist in the support of the testis, and, if well developed, assist in the closure of the inguinal canal (Ogilvie, 1937). There is a tendency to sacrifice this layer indiscriminately, but such a practice results in the loss of one of the basic mammalian protections against hernia. If it is opened carefully as a layer, and if the repair can be effected without loss of all its attachments, I am sure that it should be retained.

For the inguinal canal with adequate musculature, removal of the hernial sac is the fundamental step. Exposure of the deep inguinal ring is essential and requires adequate depth of anæsthesia so that the internal oblique can be retracted laterally. Similarly the peritoneum can only be drawn down to expose the neck of the hernial sac when the anæsthetic has relaxed the transversus. It has been a constant feature of the comparative study that the peritoneum is firmly adherent to the tendon of the transversus around the deep inguinal ring.

I must express a preference for opening the sac and ligating and transfixing its neck under direct vision, rather than blindly at the base of a twisted cord. Extraperitoneal fat may be densely adherent to the medial side of the neck of the sac. A purse-string or transfixion stitch inserted under direct vision will avoid any danger of damaging either bladder or bowel and will ensure that no pouch exists. If there is no pouch there is no need to hook the neck of the sac up under the deep surface of the internal oblique, a step that offends the liberty of the parietal layers to move upon each other.

Stitches placed into and around the deep inguinal ring can only be inserted safely if the inferior epigastric vessels are exposed and clearly seen. I submit that the conservative operation demands particular attention to the finer points of surgical technique and a critical analysis of the procedures in current use.

When the inguinal sphincter has failed or is incompetent, and in most cases of direct hernia, some form of reconstruction of the posterior wall of the inguinal canal is necessary. Whether a silk or thread lattice is employed (Ogilvie, 1937) or whether sutures of living fascia (Gallie, 1924), there is a tendency for the inguinal ligament to be split up into small ribbons of tendinous fibres. This ligament is thus easily torn where the greatest support is needed, where it is arching over the femoral vessels. Not only, therefore, is the inguinal ligament an unsound anatomical foundation for the repair, but it is proved to be disappointing in surgical experience.

An alternative to the inguinal ligament is near at hand and is regularly employed in the repair of femoral hernia. Few will deny that the classical Lotheissen's operation is a sound and effective remedy for this type of hernia. In addition to closing the femoral canal it restores the conjoined tendon to its normal insertion, the pectineal line of the pubis. It therefore restores also the posterior wall of the inguinal canal and reduces the size of the deep inguinal ring. The operation is based upon the strong unyielding fibres of Astley Cooper's ligament, a welcome contrast to the inguinal ligament.

My suggestion is not original. Mr Kenneth Heritage has been repairing inguinal herniæ for nine years by suturing the conjoined tendon down to Cooper's ligament. He referred to his method during a discussion at the Royal Society of Medicine in 1943, and will doubtless be describing it in full when his follow-up is complete. He has very kindly allowed me to watch his repair and I am satisfied that the method is sound both in theory and in practice.

The anatomy of femoral hernia is usually described separately from that of inguinal hernia and the two conditions are therefore believed to present two distinct problems. Comparative anatomy reveals that both herniæ pass-through the same basic interval, the arch of the abdominal muscles over the femoral vessels. This interval is divided superficially by the mobile medial part of the inguinal ligament and more deeply by a variable sheet of connective tissue termed the deep femoral arch (Buchanan, 1946). The mobility of this tissue dividing the interval is shown by the not uncommon appearance of a femoral hernia after attempts to perform a Bassini type of repair for an inguinal hernia. There is clearly no reason why the merits of Astley Cooper's ligament should be reserved for the repair of femoral hernia when the problem of inguinal hernia demands a strong foundation upon which reconstructive operations can be based.

~Summary and Conclusions

1. Confusion regarding the anatomy of hernia is largely due to the many recent changes in terminology.

2. Further study of the elastic tissue of the abdominal wall will elucidate the ætiology of hernia.

3. Bassini's operation has no counterpart in nature.

4. The external oblique aponeurosis is not an essential part of the mammalian inguinal ligament and is a poor foundation for repair or reconstruction of the inguinal canal.

5. There is no justification for the arbitrary separation of the anatomy of inguinal from that of femoral hernia.

6. The transversalis fascia is not one of the basic factors in the anatomy of the mammalian inguinal canal.

7. The conservative operation for hernia demands restoration of normal anatomy and often repair of the medial margin of the deep inguinal ring.

8. The conjoined tendon is attached in nature to the pectineal surface of the pubis and requires reattachment in many cases of hernia.

9. Reconstructive operations for a failed inguinal sphincter are better assured of a firm foundation if they are based on Astley Cooper's rather than on Poupart's ligament.

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THE SURGICAL PATHOLOGY OF RENAL TUBERCULOSIS

Lecture delivered at The Royal College of Surgeons of England

on

21st July, 1947

by

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TUBERCULOSIS CAUSES a great variety of lesions in the kidney and when the disease has been treated by nephrectomy the subsequent examination of the operation specimen is always an interesting procedure and often reveals something unsuspected. In most cases the gross lesions are obvious and they may even be much larger and more numerous than had been anticipated. In other cases, however, the excised kidney may at first glance look remarkably healthy and it is only when the organ has been sliced open that the tuberculous lesion is displayed. Moreover, exceptional cases are sometimes met with in which although there has been reliable pre-operative clinical and laboratory evidence of renal tuberculosis, yet the only evidence of disease in the nephrectomy specimen is a minute ulcer near one of the renal papillæ, a lesion only visible when the kidney has been carefully dissected, each calyx being opened up separately and examined with a lens. I have never yet failed to demonstrate a definite tuberculous lesion in a kidney known to be excreting tubercle bacilli, but looking back on a long experience in the examination of operation specimens I am struck by the bewildering variety of lesions revealed when tuberculous kidneys are examined. I have tried to find an explanation for this diversity and multiplicity of lesions by studying the early stages of renal tuberculosis and by tracing the gradual development of tuberculous lesions and correlating these with the results of urine analysis. These investigations have led me to distinguish four stages in the development of the lesions of renal tuberculosis, the recognition of which enables one to build up a clearer mental picture of the progress of the disease. The four stages I shall describe are, of course, only passing phases of a continuous process, and to see them in their proper setting we must first recall the source of infection and the general development of tuberculous lesions in the kidneys.

Source of Infection

In most cases of renal tuberculosis there is no doubt that the infection reaches the kidney by the bloodstream, being transported there from a tuberculous lesion in some other part of the body. Occasionally, perhaps, infection may spread to the kidney by the peri-ureteral lymphatics and an ascending infection by the ureter is a possibility in cases of advanced tuberculosis of the bladder accompanied by obstruction. In most cases, however, the renal infection is obviously derived from a primary focus

in the lungs or from an active tuberculous lesion elsewhere. Only hæmatogenous infections are now being considered.

Initial Tuberculous Lesions

The initial lesion caused by a hæmatogenous infection is rarely seen, but when met with accidentally is found to consist of a cluster of small discreet greyish-white tubercles scattered throughout the kidneys, but most numerous in the cortex. This kind of lesion does not give rise to any signs or symptoms of renal disease but may be accompanied by a tuberculous bacilluria, though at this stage tubercle bacilli are only excreted intermittently and in small numbers.

Further Development of Tuberculous Lesions

The further development of a tuberculous lesion in the kidney depends partly on the virulence of the bacteria, and partly on the resistance of the patient, but the actual situation of the lesion is also of considerable importance. The significance of the immunity factors has been made clear by the work of Medlar (1926) who showed that if an individual has a high resistance to tuberculosis, then caseation does not occur, but a hyperplastic type of lesion develops, resulting in the destruction of the tubercle bacilli and leaving only a small scar. On the other hand, if the patient has less natural resistance a different type of lesion develops, characterized first by cellular infiltration, and, later, by areas of necrosis. If this necrotic lesion is so situated that it can discharge into the renal pelvis, then ulceration and cavitation will follow.

The anatomical arrangement whereby the collecting tubules unite to discharge at the renal papillæ is a most important consideration in relation to the extension of the disease within the kidney. If infectious material should pass down the convoluted tubules then fresh tuberculous lesions tend to form at the site of convergence of the collecting tubules. The coalescence of these microscopic lesions results in secondary foci near the apex of papillæ. These lesions are small but of special importance, because if a tuberculous ulcer has formed in a renal calyx it tends to eat its way back into the kidney, resulting in a cavity lined with yellow caseous material. Thus, cavities may arise either from the breaking down of lesions in the kidney, or from the extension of ulcers in the pelvis. The later development is determined, to a large extent, by the degree of fibrosis and obstruction which may follow further extension of the disease to the renal pelvis, ureter and bladder.

Tuberculous Lesions Found in Nephrectomy Specimens

In cases of renal tuberculosis treated by surgeons the commonest lesions to be found are (1) cavities lined with shaggy granulations, often with a sinus discharging into the renal pelvis ; (2) round caseating nodules embedded in apparently healthy surviving kidney tissue ; (3) small granulations on minute ulcers in the calyces or in the mucous membrane of the

pelvis. If there has been much obstruction in the renal pelvis or ureter there may also be a variable degree of pyonephrosis, fibrotic atrophy and increased deposition of fat. In more advanced cases large tuberculous cavities become filled with inspissated putty-like necrotic material, in the walls of which calcium salts may be deposited.

These are the commonest lesions found in surgical tuberculosis and as a rule they are recognisable at a glance, though occasionally atypical tuberculous lesions are met with which might be mistaken for something else. For instance, the word tuberculoma has been used to describe tuberculous lesions which resemble a tumour, and a remarkable case of this kind has been described by Bugbee (1941). The patient was a nurse, aged 30, who was operated on for tuberculosis of the left kidney because pus and tubercle bacilli had been found in the urine from that side, but when the kidney had been removed it showed the renal pelvis to be filled with a smooth, pinkish, firm mass resembling a renal tumour. It so happened that the operation had been carried out before a group of twenty-two experienced urologists, most of whom when they had looked at the operation specimen were of the opinion that a mistake had been made in the diagnosis of renal tuberculosis and that the pathological lesion present was actually a pelvic tumour. However, microscopic examination subsequently proved it to be a tuberculous lesion. Bugbee states that in a search of the literature he had been unable to find a report of a similar case, but a few other cases closely resembling this have since been reported.

This brief review of the origin and development of tuberculous disease of the kidney brings into prominence the variable character and extent of the lesions so that it would seem to be a particularly difficult task to take an operation specimen exhibiting a fairly advanced lesion and to reconstruct and visualise its development. These difficulties are accentuated by the fact that " tuberculosis obliterates the very pathways along which it has advanced " (Lieberthal, 1938). Nevertheless, by working through a large series of cases and correlating the clinical, bacteriological and pathological features, we can piece together the whole story from beginning to end. Having attempted to do this it now seems clear to me that the story can be divided most naturally into four chapters, which I have called the four stages in the development of renal tuberculosis. The recognition of these adds greatly to the interest of examining a particular case, and I venture to think that the subdivision of the disease in this way may be of clinical interest also, especially in relation to the problems of tuberculous bacilluria and the possibility of natural cure in the early stages of the disease.

The Four Stages in the Development of Renal Tuberculosis
STAGE 1.

Tubercle bacilli are brought to the kidneys by the bloodstream, being derived from a tuberculous lesion in some other part of the body, and

being deposited in the kidney they give rise to multiple microscopic lesions situated chiefly in the cortex (Fig. 1). These lesions do not cause any urinary symptoms or betray their presence in any noticeable way. The distinctive features of this first stage of renal tuberculosis are that the lesions are multiple and bilateral and that they cause no symptoms. Also at this stage the urine does not contain pus or blood, but a trace of albumen is generally found, a point which will be referred to later. Tubercle bacilli are excreted intermittently and in small numbers and are only likely to be detected by repeated guinea pig or culture tests.
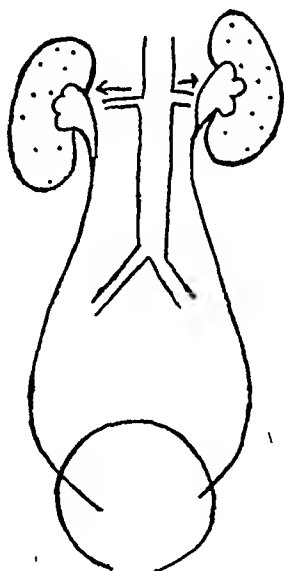
Fig. 1.
FIRST STAGE OF RENAL
TUBERCULOSIS
Hæmic emboli of tubercle bacilli
causing bilateral multiple micro-
scopic lesions.
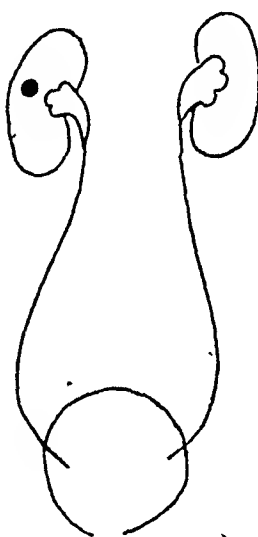
Fig. 2.
SECOND STAGE OF RENAL
TUBERCULOSIS
One of the tubercles has
developed into a visible lesion.
The others have disappeared.
Tubercle bacilli now only
occasionally present in urine.

STAGE 2.

The majority of the lesions in the first stage heal spontaneously, as do tuberculous lesions in other organs of the body, but one or two may persist and increase in size, causing lesions visible to the naked eye. A Stage 2 lesion is represented in Fig. 2 as a solitary surviving and expanding lesion embedded in one kidney. Renal tuberculosis at this stage also causes no urinary symptoms, though, as before, it may give rise to transient bacilluria. If the solitary lesion can be encircled by fibrous tissue there is still a chance of arrested development, or even of natural cure, but it is more common for it to progress in the way about to be described.

STAGE 3.

The third stage is reached if the slowly growing isolated lesion in the kidney breaks down and discharges into the renal pelvis (Fig. 3). This alters the whole character and course of the disease. In other parts of the body the breaking down of an abscess and discharge of its contents is a step towards healing. In the kidney the exact opposite is the case. The material evacuated into the renal pelvis consists of cellular débris and living tubercle bacilli which spread the infection, first to the renal pelvis and ureter, and, later, to the bladder. Now, for the first time, the patient begins to complain of urinary symptoms and the urine is found to contain pus cells, red blood corpuscles and tubercle bacilli in sufficient numbers to be found fairly easily in stained films. There is now no longer any prospect of natural cure and, if the disease is unilateral, nephrectomy is the best treatment.
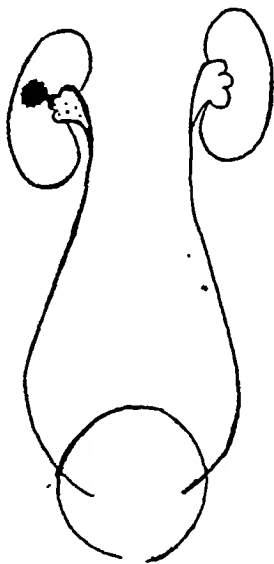
Fig. 3.
THIRD STAGE OF RENAL
TUBERCULOSIS
Discharge of tubercle bacilli down renal tubules leads to to tuberculous ulcer at apex of papillæ. Tubercle bacilli now constantly present in urine. Little chance of natural cure.
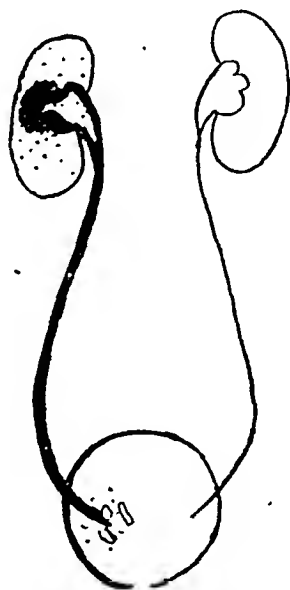
Fig. 4.
FOURTH STAGE OF RENAL
TUBERCULOSIS
Tuberculous cavity communicating with renal pelvis. Lesions appearing in ureter and bladder. No hope of natural cure. Nephrectomy indicated.

STAGE 4.

The fourth stage is the result of extension of the disease to the renal pelvis, ureter and bladder (Fig. 4). It is characterized by the development of secondary tuberculous lesions elsewhere in the kidney, by tuberculous ulceration and fibrosis of the ureter, and by the appearance of tuberculous ulcers in the bladder. This is the stage of development most commonly found when nephrectomy is performed. If the disease is allowed to continue it may, of course, spread still further in the urinary and genital

organs, gradually undermining the patient's health. In some cases, however, the further progress of the disease is a surprisingly slow process and it is never safe to prophesy how long its course may be.

Most hospital museums are well supplied with operation specimens illustrating large tuberculous cavities, pyonephrosis and fibrotic atrophy secondary to tuberculosis, but the earlier stages of the disease which are really the most instructive to the student are seldom represented. The tuberculous lesions which I have described as Stage 1 and Stage 2 are naturally rarely encountered because they do not cause symptoms and would, therefore, only be chance findings at autopsy or when nephrectomy has been performed for some other purpose. The first stage of the disease is most likely to be found in patients dying fairly early from pulmonary or bone or joint tuberculosis. The second stage lesion may sometimes be found in association with more advanced stages, as in the kidney illustrated in Fig. 5. This was a nephrectomy specimen, in the upper part of which two tuberculous lesions were found. One had discharged into the renal pelvis and this was the source of the tubercle bacilli found in the urine. This lesion appears in the photograph as a dark area with ill-defined margins in the upper calyx (Stage 3 lesion). Close by this (marked with an arrow) is a small, round tuberculous lesion embedded in the kidney (Stage 2).

Fig. 5. At the upper pole of the kidney two tuberculous lesions are present, one of which has discharged into the upper calyx, whereas the other has not.

Both Stage 2 and Stage 3 are represented also in Fig. 6, which is a photograph of a tuberculous kidney with double renal pelvis and double ureter. Before operation pus and tubercle bacilli were found in the urine collected from the upper renal pelvis with which a tuberculous cavity communicates (Stage 3). A closed tuberculous lesion (Stage 2) is also present at the lower pole of the kidney, but this had not discharged into the lower renal pelvis, and urine from the lower ureter was free from pus and no tubercle bacilli were found in films. If the disease had been allowed to continue the lesion in the lower pole of the kidney would soon have progressed from Stage 2 to Stage 3.

Fig. 6. Tuberculous kidney with double renal pelvis and double ureter. An " open " tuberculous lesion (Stage 3) communicates with the upper ureter from which pus and tubercle bacilli were recovered before operation. The lesion in the lower pole is still closed and does not communicate with the lower renal pelvis (Stage 2) and no pus or tubercle bacilli were found in urine from the lower ureter when catheterised before operation.

In describing the course of tuberculous lesions in the kidney I have assumed that healing is a natural and common process in the first stage of renal tuberculosis, and also that tuberculous bacilluria can occur at a pre-clinical or occult phase of the disease. Misunderstandings have arisen in the past with regard to both these questions, but some of these can be removed by defining more precisely the possibilities and limitations of

natural healing. So I will attempt to do this and also explain the significance of tuberculous bacilluria in relation to the four stages of the disease which have been outlined.

The Possibility and Limitation of Natural Cure in Renal Tuberculosis

In the advanced tuberculous lesions with which the surgeon is familiar there is, of course, no possibility of a natural cure. But it is important to remember that in its earlier stages a tuberculous lesion in the kidney may heal spontaneously. Natural healing very rarely occurs if the tuberculous lesion in the kidney has progressed far enough to cause urinary symptoms, but it is easy to prove that in its occult or pre-clinical phase tuberculosis of the kidney may heal completely. Sanatorium patients, for instance, with no signs or symptoms of genito-urinary tuberculosis may excrete tubercle bacilli in the urine, and if such patients die, small tuberculous foci are invariably found (if looked for) in the kidney. On the other hand, if these patients improve in their general health, they cease also to excrete tubercle bacilli in the urine, from which it may be inferred that the early sub-clinical tuberculous lesion in the kidney has now healed.

Strong support for this point of view is supplied by the work of Band (1943) who has continued for many years a research into the incidence of tuberculous bacilluria in sanatorium patients showing no clinical signs or symptoms of urinary tuberculosis. Like other observers he found that such patients often excreted tubercle bacilli in the urine, and if they died, minute tuberculous lesions at early stages of development could be found in the kidney. The earliest lesions were found in relation to a glomerulus or in a capillary between the tubules close to a glomerulus. Evidence of healing was shown by the fact that some follicles with central epithelioid cells were surrounded by spindle cells, whereas in others the central area had been hyalinised and the amorphous caseous-like débris had been walled off by organizing fibrous tissue cells. In other lesions nothing remained but scar tissue.

Baggenstoss and Greene (1941), who have also made a study of the process of healing in renal tuberculosis point out how difficult it is to decide the question by histological examinations alone. They observe that it has sometimes been said of renal tuberculosis that if the histological diagnosis is possible the disease has not healed, and on the other hand, if it has healed, it can no longer be recognised as tuberculosis ! These are not their own views because they collected fifteen cases of healed or healing tuberculous lesions in the kidney. These were all patients who had had tuberculosis and had died from other causes. In most instances there were signs of healed or regressing tuberculous lesions in other organs beside the kidney. They concluded that healing of tuberculosis does occur, but only in the early stages of a renal lesion. Once the disease has reached the stage of causing symptoms, or clearly demonstrable changes, then it rarely, if ever, heals.

The limitations of natural healing have been well defined by Auerbach (1940), who has pointed out that in tuberculous disease of other organs, when extensive destructive changes occur, healing is only possible by a process of encirclement of the lesion and ultimate encapsulation by fibrous tissue. For example, in pulmonary tuberculosis a caseous focus may become surrounded by a fibrous capsule, and eventually become calcified, but such an encirclement with fibrous tissue is not possible in a renal tuberculous lesion which is in open communication with a renal calyx and over which urine is continually being passed. Thus we reach the rather paradoxical conclusion that natural and spontaneous healing can only occur in those tuberculous lesions of the kidney which have never caused urinary symptoms. Once the disease has progressed to the stage of causing urinary symptoms we may assume that an open lesion exists for which there can be no expectation of spontaneous or natural cure.

The Significance of Tuberculous Bacilluria

There is now overwhelming evidence that when tubercle bacilli are found by guinea pig or culture tests in the urine of patients free from any urinary symptoms, but suffering from tuberculosis of the lungs, bones or joints, these tubercle bacilli are derived from minute tuberculous lesions in the kidney. The " excretion without lesion " hypothesis has been contradicted by almost everyone who has used the experimental method or checked conclusions by careful post-mortems. One of the most thorough investigations in recent years is that carried out by Ordway and Medlar (1942) who followed up 287 patients with clinically active pulmonary tuberculosis and 112 patients without evidence of pulmonary tuberculosis over a period of ten years. They found that tubercle bacilli were sometimes, but not frequently, excreted in the urine of patients suffering from pulmonary tuberculosis but free from urinary symptoms. They showed also that though the method of random sampling only rarely showed the presence of tuberculous bacilluria, the excretion of tubercle bacilli in the urine could be proved in a much larger number of cases by repeated tests on larger volumes of urine than are generally employed. Tuberculous bacilluria is a possibility in all cases of pulmonary tuberculosis; the frequency with which it can be demonstrated depends on the methods adopted.

Albuquerque, Paz and Magard (1943) have also supplied evidence that patients in the terminal stages of chronic pulmonary tuberculosis eliminate tubercle bacilli through the kidneys, although they may not complain of any urinary symptoms. The urine of thirty patients was examined daily by films, cultures and guinea pig tests over a period of several weeks ; in most cases, in fact, until death supervened. The conclusion to be drawn from this careful investigation is that tuberculous bacilluria is quite common in association with pulmonary tuberculosis and that it may occur in the absence of urinary symptoms.

Reference should also be made to the work of Rosencrantz (1940) who examined the urine and genital organs in 300 proved cases of chronic pulmonary tuberculosis. None of these patients complained of urinary symptoms, but tubercle bacilli were found in the urine by guinea pig tests in fourteen cases.

In tuberculous bacilluria unassociated with symptoms of urinary disease the tubercle bacilli are rarely sufficiently numerous to be found in stained films of a urine deposit and can only be detected by guinea pig tests or cultures.

A routine weekly examination of the urine for albumen is a simple and reliable method of selecting cases to test for tuberculous bacilluria. Colby (1940) found a surprisingly high percentage of positive guinea pig tests in patients in whose urine a small amount of albumen was noted for the first time. Some of these patients were later found to have pus cells and red blood corpuscles in the centrifuged deposit, but the guinea pig tests for tubercle bacilli were positive before these appeared, when the only abnormality detected by urine analysis was a slight albuminuria.

We are now in a position to group together the features which characterize the excretion of tubercle bacilli in tuberculous bacilluria. They are as follows : (1) Only a few tubercle bacilli are excreted in the urine and it is very rare for them to be discovered in stained films of the centrifuged deposit. Guinea pig inoculation is nearly always necessary for the demonstration of tuberculous bacilluria. (2) The excretion of tubercle bacilli is intermittent, so that the test may be positive on one occasion and negative on another. (3) The urine is clear in appearance and does not as a rule contain pus or blood, but at the time when tubercle bacilli are being excreted the urine generally contains albumen. (4) In cases of tuberculous bacilluria the excretion of tubercle bacilli may cease when the patient's general health improves (Dukes, 1939).

Summary

This review of the surgical pathology of renal tuberculosis includes a description of four stages in the development of tuberculous lesions in the kidney. Stage 1 is the result of a hæmatogenous infection derived from a tuberculous lesion elsewhere in the body and is characterized by multiple microscopic lesions situated chiefly in the cortex. These lesions do not cause any symptoms though they may be accompanied by slight albuminuria and a transient tuberculous bacilluria. Most of these lesions heal spontaneously and completely.

Stage 2 is the result of the further growth of a surviving Stage 1 lesion and takes the form, as a rule, of a solitary, round greyish-white tuberculous focus embedded in the kidney. This also causes no urinary symptoms, though it may be accompanied by albuminuria and transient tuberculous bacilluria. If this lesion can be encircled by fibrous tissue there is still a chance of natural cure, but most of these lesions continue to develop to Stage 3.

Stage 3 is reached when the tuberculous lesion embedded in the kidney breaks down and discharges into the renal pelvis. This results in the extension of the disease to the renal pelvis and ureter. Now, for the first time, the patient begins to complain of urinary symptoms and the urine is found to contain pus cells, red blood corpuscles and tubercle bacilli in sufficient numbers to be found fairly easily in stained films of the centrifuged deposit.

Stage 4 is characterized by the development of secondary tuberculous lesions elsewhere in the kidney and by further extension to the ureter and bladder. This is the stage of development most commonly found when nephrectomy is carried out for the treatment of renal tuberculosis. The further development of tuberculous lesions in the kidney beyond this stage is determined chiefly by the extent of fibrosis and obstruction.

The possibilities and limitations of natural healing and the significance of tuberculous bacilluria are more easily understood if the course of the disease is divided into these four stages.

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THE RESTORATION AND DEVELOPMENT FUND

SINCE THE REPORT on the Fund in the April number of the ANNALS the following notable gifts have been received:—Mr. Job Wild, friend and former patient of the President, made the most generous gift of £2,500, which, with the £500 he had given previously, makes his total contribution £3,000. Five hundred pounds has been received from Messrs. Mersons (Sutures) Limited, and one hundred guineas from J. I. Munro Black and Thomas Moore.

These gifts bring the total received to £189,500.

HER MAJESTY QUEEN MARY

HER MAJESTY QUEEN MARY graciously honoured the College by a visit on 22nd May. After the Members of the Council and the Officers of the College and their ladies had been presented, Queen Mary inspected the Museum including the newly restored Hunterian Collection. Her Majesty was greatly impressed by the progress which had been made and expressed approval of the plans for rebuilding and enlarging the College. Mr. Arthur Sims who endowed the Commonwealth Travelling Professorship had fortunately just returned from New Zealand, and he and Mrs. Sims were present on the occasion. Queen Mary expressed great appreciation of Mr. Sims's generous gift which Her Majesty thought would not only advance the science of medicine, but would be a valuable Imperial link. Her Majesty then took tea in the Library.

By kind permission of The " Topical " Press Agency Limited.

Her Majesty Queen Mary sees the statue of John Hunter relieved of the brickwork cover which completely protected it from enemy action.

Her Majesty Queen Mary inspecting the
bricked-up statue of John Hunter in July, 1945.

THE LIBRARY

DURING RECENT MONTHS the historical collections in the library have
received further valuable accessions by gift and purchase—the most
important is a splendid copy of the copper-plate engravings of Vesalius'
anatomical plates edited by Jacques Grévin and printed at Paris in 1564.
Vesalius' original plates were printed from wood-block engravings in
1543, and to his annoyance were quickly copied in England by a skilled
craftsman in the new process of engraving on copper. These were pub-
lished, with full acknowledgment to Vesalius, in 1545 by Thomas
Geminus. The library has long possessed a copy of Geminus' edition
which came from Sir Anthony Carlisle, President in 1828. Geminus
appears to have been a prótégé of the Crown. The various editions of his
book are dedicated to Henry VIII who died in 1545, to Edward VI who
died on July 6, 1552, while the second edition (1553) was in the press, and
to Elizabeth in the second year of her reign 1559. The young French
physician and scholar Jacques Grévin is known to have been at Elizabeth's
court about this time and Dr. Cushing in his great study of Vesalius
suggests that Grévin himself arranged the purchase of Geminus' copper-
plates for the Paris publisher André Wechel. At all events in 1564 Wechel
issued this noble edition of the Geminus-Vesalius plates with additional

commentary by Grévin. The book is of considerable rarity—Cushing recorded only seven copies and only one, the British Museum copy, in this country.

Wechel was the publisher of Jean Fernel, the father of physiology, and two more editions, each of some textual importance, have been added to the series of Fernel's works in the library; both are of his collected *Universa medicina*, that of 1574 was one of the earliest books issued by Wechel after his exile, as a Protestant, from Paris to Frankfort, and is the first edition of Fernal to contain the letter from Crato von Craftheim, physician to the Emperor, in which he speaks of Fernel as the supreme authority in contemporary medicine; the other new acquisition, the Genoa edition of 1578, is the first to contain Fernel's treatment of fevers.

Three earlier surgical texts have been added to the library. First, the standard edition by Joseph Pétrequin of Hippocrates' Surgical works, with elaborate commentaries, published in Paris 1877-78, which is an essential book for any study of ancient surgery. Secondly, an " incunabulum," the *Chirurgia* of Pietro d' Argelata, Venice 1497. Argelata was Gui de Chauliac's most distinguished pupil and a pioneer in the use of sutures and drainage-tubes in the treatment of wounds. The third is an early edition (1571) of the collected works in medicine and surgery of Leonardo Fioravanti, an Italian surgeon, whose teachings were followed throughout Europe in the sixteenth century. With these may be recorded two other books by Italian writers, both on the treatment of venereal disease : Benedetto Vettori's *De Morbo Gallico*, Florence 1551 and Leonardo Botallo's *Luis venereae curandae ratio*, Paris 1563. Vettori was a prominent humanist, professor successively at Padua and Bologna ; Botallo an Italian surgeon practising at Paris, renowned for his treatment of gunshot wounds.

The seventeenth-century collection has been enriched with two small but important English works. James Cooke's *Supplement to the Marrow of Chyrurgerie*, London 1655, is a very much rarer book than the text which it supplements and of which the College already possesses copies. Cooke practised at Warwick and translated the Latin writings of Dr. John Hall of Stratford, Shakespeare's son-in-law. John Moyle's *Chirurgus marinus or the sea chirurgion*, London 1693, is a landmark in the literature of British naval surgery. The book appears to be founded on the author's own experiences. He had served in the last Dutch wars in Charles II's reign and seems also to have known all the Mediterranean shores and to have crossed the Atlantic to Newfoundland.

Next in interest to the Vesalius, described at the beginning of this paper, are the accessions to the eighteenth-century books. Mr. Ernest Finch, F.R.C.S., has presented a copy of the prize essay on Cancer (*De cancro*, Paris 1774) by Bernard Peyrilhe. This essay won the prize offered through the Dijon Academy of Medicine by Claude Pouteau, surgeon to the Hôtel-Dieu at Lyons, and one of the most distinguished surgeons

of the day. Peyrilhe's views, though in fact quite unscientific, roused considerable interest, and his little book was translated into French, German, Dutch and English. This original Latin edition is a rarity : only one other copy has so far been traced in London, at the Wellcome Library, and it is not in the major American medical libraries. Peyrilhe at the time of writing it was a young surgeon in Paris, he later became Professor of chemistry in the schools of the Royal Academy of Surgery, and after the Revolution was the first professor of materia medica in the new Ecole de Santé. He was a man of versatility, wrote a history of surgery, and suggested that the pharmacopœa should be based on native products rather than rely so largely on imported drugs.

Antonio Campani's *Odontologia*, Florence 1786, of which a copy has been bought, is the classic Italian contribution to the evolution of dentistry, particularly valuable for its illustrations of dental instruments. It is a peculiarly fit acquisition, at the time when the dental section of the library is being developed for the new Faculty of Dental Surgery. Similarly fortunate in connexion with the formation of the Faculty of Anæsthetists is the gift from Mr. E. F. Storey of letters between James Moffat and Matthews Duncan concerning Sir James Simpson's experiments with anæsthetics.

Two unusual books have been presented on permanent loan by Professor Lambert Rogers, F.R.C.S. : John Innes, *Eight anatomical tables*, Edinburgh 1776, and an American edition (Philadelphia 1833) of Sir Charles Bell's *Engravings of the arteries*. The College possesses Bell's original drawings for this important work as well as the first printed edition (1801).

Another important Americanum, this time a British edition of an American classic, which has been added to the library, is a beautiful copy in original boards of Benjamin Rush's *An account of the bilious remitting yellow fever as it appeared in the city of Philadelphia in the year* 1793, Edinburgh 1796. Rush, who was the leading physician in Philadelphia, besides being an eminent teacher and savant and a strong willed politician who had signed the Declaration of Independence and later deserted Washington, endangered his own health in his heroic fight against the epidemic. The College possesses a copy of the first edition of his graphic record of the disease, published at Philadelphia in 1794; the Edinburgh re-issue is an even rarer book.

ANÆSTHESIA FOR THORACIC SURGERY

Lecture delivered at The Royal College of Surgeons of England

on

8th October, 1947

by

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IT IS SAID that an ounce of practice is worth a pound of theory and in this lecture I will confine myself to methods of anæsthesia for some typical thoracic operations which I have myself found satisfactory. I am, of course, not suggesting that other methods do not give equally good results.

Drainage of Acute Empyemata

These operations are usually performed quite satisfactorily under local analgesia. This presents no difficulty if some of the solution is injected beneath the periosteum of the rib. Severe wound infection may occur after the drainage of acute streptococcal empyemata unless it is remembered that the action of the sulphonamides is inhibited by minute accounts of local analgesics containing the p-amino benzoic acid grouping. Nupercaine does not come into this category and is suitable as a local analgesic in a dilution of about 1 in 1,500, with adrenaline added in the proportion of 1 in 250,000.

Thoracoplasty

Thoracoplasty is an operation which as time goes on appears to last longer and longer. Twenty years ago a complete thoracoplasty was often performed in 20 minutes and no special anæsthetic problems arose. Nowadays, however, with a much more extensive rib removal, the operation is usually done in three stages, the first sometimes taking nearly two hours and the others from one to one-and-a-half hours.

There is a sharp divergence of opinion among thoracic surgeons as to the relative merits of local and general anæsthesia. As one who has worked with both types of surgeon I think I can claim to be unbiased in this matter, and in my view, each case must be considered on its merits. Some patients are temperamentally unsuited for any form of local analgesia, and would much prefer to be unconscious. Furthermore, in my experience, the operative upset as judged by a study of the T.P.R. charts over a 24-hour period is, on the average, greater after local than after general anæsthesia. The stock argument brought against general anæsthesia for such cases is that as the lungs are diseased they should not have to cope with anæsthetic vapours. This has always seemed to me to

be fallacious and can easily be countered as follows: most physicians would agree that a patient with active phthisis would benefit temporarily from mild oxygen therapy (say 80 per cent. oxygen, 20 per cent. nitrogen) combined with shallow respiration. Suppose that for the nitrogen we substitute another non-irritating gas which is a slight respiratory depressant but which happens to have narcotic properties such as cyclopropane, we have an anæsthetized patient who is breathing shallowly with a high oxygen content in the inspired mixture. It is difficult to see any objection on this score. On the other hand, patients with extensive cavitation and copious sputum usually do best with local analgesia.

The technique for *general anæsthesia* which I now use is induction by intravenous thiopentone followed by cyclopropane usually combined with minimal curare. As relaxation is not essential, it might be thought strange to use curare in thoracic surgery. In practice it is found that the drug facilitates a very smooth anæsthesia and the percentage of cyclopropane can be so much reduced that cardiac arrhythmias are uncommon. Incidentally I am not enamoured with the current idea that cardiac irregularities only occur during a particular plane of cyclopropane anæsthesia, and that " pushing " the drug will restore a normal rhythm. I do not believe that this practice is sound. Tracheal intubation is not essential in dry cases but should be used if there is an appreciable amount of sputum so that suction can be used without delay if necessary.

If, on the other hand, it is decided to perform thoracoplasty under *local analgesia*, I employ a method modified from the Brompton Hospital technique. For a first stage (upper) thoracoplasty a brachial plexus block is first performed by the usual supraclavicular route. This facilitates scapular retraction by relaxing the muscles. A wide and deep infiltration of the line of incision is next undertaken, taking particular care to inject plenty of solution under the posterior border of the scapula. Finally, five subcuticular wheels are raised for the reception of the towel clips. I do not do a preliminary paravertebral block as I believe it is better for the surgeon to block the intercostal nerves under vision as the operation proceeds. This can be done with greater precision and with the expenditure of less solution. An adequate local analgesia for thoracoplasty is a fairly formidable procedure and at least half an hour should be allowed for it. I use one solution throughout as no mistakes can then be made as to dilutions and rather more latitude is possible as to the total volume injected. If double-strength solution is used for the nerve blocks, toxic reactions are definitely more common. The actual mixture I use is equal parts of 1 in 2,000 amethocaine and 0.25 per cent. procaine with 1 in 500,000 adrenaline made up with saline into an isotonic solution. This is done as follows:

Sodium chloride	4·45g.	
Procaine	1·25g.	are dissolved in 500 c.cm. of double-
Amethocaine	0·25g.	

315

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distilled water. This is sterilized by boiling and after being cooled, 1 c.cm. of 1 in 1,000 adrenaline is added from an ampoule. This combined solution gives a rapid onset of analgesia and a fairly prolonged effect (usually up to 2½ hours). The use of a continuous-flow syringe for the infiltration obviates constant refilling and saves time.

The chief disadvantages of the analgesic technique which I have described are the time necessary and the multiple injections which are very trying for the patient. In order to obviate these, various alternatives have been tried. About ten years ago I did a series of 30 cases using a *unilateral high spinal block* with light nupercaine. Although I did not have any serious trouble, the margin of safety was too small and I have now abandoned the technique.

From a series of 60 cases recently reported by Durrans, it would seem that the *extra-dural spinal block* method has great possibilities. The injection into the peri-dural space is not made in the lumbar region but at the site of election. The spinal needle is advanced until an area of negative pressure is encountered and from 20 to 25 c.cm. of solution are injected. This solution consists of 0·1 per cent. amethocaine, 0·5 per cent. procaine in 0·84 per cent. sodium chloride. The resulting analgesia extends over 4 to 6 segments above and below the site of the puncture. If on further investigation the favourable results are confirmed the extra-dural spinal technique may well prove very useful for thoracoplasty as most of the disadvantages and dangers of the sub-arachnoid method are eliminated.

Before leaving the subject of thoracoplasty it must be remembered that the operation leaves the patient with a mobile chest wall and it is most important that the affected side should be supported and immobilized with strapping. Failure to carry this out may result in partial paradoxical respiration with rapid deterioration of the patient's condition.

There are two further points about thoracoplasty which do not directly concern the anæsthetic but which are nevertheless important. First, this operation is not tolerated at all well by the middle-aged and old patient. In my experience, the ultimate prognosis in patients over 50 is extremely poor. In the second place, I think that we should be very critical of the slightest upper respiratory infection or of any rise in temperature above the patient's normal level in the period immediately preceeding operation. Only too often a " cold " is a euphemism for an exacerbation of the tuberculous focus and surgical intervention at this stage may well end in disaster. A skiagram of the chest taken the day before operation and compared with previous ones may help to clear up this point.

Pulmonary Lobectomy and Pneumonectomy

These operations are unsuitable for local analgesia and it is now generally agreed that the anæsthetic technique of choice is endotracheal cyclopropane using controlled respiration when desirable. The operation

involves the opening of one and occasionally both pleural cavities and there is no doubt that controlled respiration is the most efficient means of ensuring adequate pulmonary ventilation and at the same time avoiding mediastinal flap. The tracheal tube should be the largest that will comfortably pass the glottis and for this reason oral intubation should be performed with a direct-vision laryngoscope. Minimal doses of curare are helpful, not only for decreasing the necessary percentage of cyclopropane as mentioned before, but also in controlling the annoying cough reflex which may occur during dissection of the hilar region even if this area has previously been infiltrated with procaine solution. The only serious drawback of this technique is that the diathermy cannot be used inside the pleural cavity although with a really gas-tight circuit it is customary to allow this for sealing vessels of the thoracic wall. Most surgeons are willing to forgo the diathermy for the perfect operating conditions which cannot be obtained with any other known method of anæsthesia. I might mention in passing that it is possible to use a non-inflammable mixture of cyclopropane, helium and oxygen, but the cyclopropane percentage is so small that a light narcosis only is obtained while the oxygen is not always sufficient to avoid anoxia in thoracic surgery. The method, though ingenious, is therefore not very practicable.

An intravenous drip should always be set up at the beginning of a thoracotomy and saline, glucose, plasma or blood given as dictated by the patient's condition. The blood-pressure and pulse readings are of interest but too much weight should not be given to low pressures. On several occasions I have observed patients so collapsed that no readings at all could be taken for over half an hour and yet the patient gradually rallied during closure of the chest wall and ultimately recovered. There is a real danger, particularly in malignant cases, of the anæsthetist stopping the operation and the patient thereby being deprived of his last chance of cure.

At the end of the operation, the surgeon may like the remaining part of the lung to be expanded so that the resultant pneumothorax is minimised. This can quite easily be accomplished by manual pressure on the rebreathing bag. This simple movement is also all that is required for maintaining controlled respiration. The various spiro-pulsators which have been designed to replace the rebreathing bags on gas-oxygen machines are complicated and in my opinion, unnecessary.

Removal of Mediastinal Tumours

The techniques already described for lobectomy and pneumonectomy also apply to tumours outside the lung, e.g., malignant retro-sternal goitres. Sometimes, however, these press on or distort the trachea to such an extent that considerable dyspnœa is present. In this event, it is wise to avoid an intravenous barbiturate and the patient should first be placed in the position in which breathing is least embarrassed. This usually involves considerable flexion of the head. Pure oxygen is then

given and cyclopropane added slowly and carefully until narcosis is established. A laryngoscope is then passed and a fairly stiff tracheal tube is introduced until its distal end is definitely beyond the obstruction. This manœuvre may have to be carried out rapidly, as the extension of the head may cause complete respiratory obstruction. In my opinion it is a mistake to attempt blind nasal intubation, as failure may result in severe laryngeal spasm and in any event it is difficult to pass a sufficiently stiff tube through the nose without trauma.

In cases of extreme respiratory obstruction the addition of helium to the mixture of cyclopropane and oxygen is invaluable as a temporary measure until intubation can be carried out, and on one occasion, in my experience, it definitely saved the patient's life.

" Wet-Lung " Operations

The removal of part or the whole of a lung for bronchiectasis is one of the most difficult operations with which the anæsthetist has to deal. It may as well be admitted at once that no really satisfactory technique has yet been evolved.

Many ingenious methods have been worked out to dam up the pulmonary secretions and evacuate them by suction, but these all depend on cuffed tubes or packs. After some experience with these devices, I have come to distrust them most profoundly. In the first place, it takes a great deal of practice to be certain that one can place a cuff or pack in one particular spot, especially when vision through a bronchoscope is obscured by bubbles of foul-smelling pus. Secondly, assuming that the cuff or pack is exactly in the right position, it may move, either because of temporary coughing, or from the manœuvres of the surgeon inside the chest. Lastly, when a cuff has been inflated it must remain with the same degree of distension throughout the operation or at any rate until the need for it has passed. I regret to say that close inspection of cuffed tubes at present available in this country does not inspire this confidence and, as a matter of fact, I have several times had them deflate while in situ. It is true that the pilot balloon may give an indication of failure but it may prove impossible to re-inflate them. The bursting of a cuff is attended with a definite risk and a death from this cause has been reported. The balloon on the Thompson tube has a silk mesh cover to prevent bursting, but I do not think that the consequent rough surface is good for the bronchial mucosa. For these reasons, I have come to believe that an anæsthetic technique using gravity drainage is preferable to one dependent upon artificial blocks in the respiratory passages, even if in theory the latter may show some advantages.

A simple and reliable method is that first described by Dr. Nosworthy. Careful postural drainage is first carried out and if there is reason to believe that an appreciable amount of secretion still remains in situ, a suction bronchoscopy is undertaken. After the induction of general anæsthesia,

usually by means of intravenous thiopentone and cyclopropane, a long, uncuffed tracheal tube is passed through the mouth and anæsthesia is maintained by cyclopropane given by a face-piece over the tube. Drainage is encouraged by a steep Trendelenburg tilt and intermittent tracheal suction can be used if necessary at any stage of the operation. The principle of the technique is familiar to most anæsthetists by the illustrations of Dr. Nosworthy's paper, but a few practical points should be noted:

(1) The largest uncuffed tube which will pass the glottis without trauma should be used.

(2) The tube should be about three inches longer than is usual and should be threaded through a London Hospital pattern oral prop to avoid kinking by the teeth.

(3) The tube should be brought out through the special face-piece designed by Dr. Morton if one is available. A gas-tight fit is secured by means of a sorbo rubber washer and the whole assembly is strapped on the face by means of a rubber harness.

(4) The Trendelenburg tilt must be a steep one, i.e., nearly 45 degrees, and the surgeon must be willing to co-operate in this. The lower part of the operating table can be horizontal if a split table is used.

Thymectomy

The operation of thymectomy for the relief of myasthenia gravis has been introduced fairly recently, and presents some problems for the anæsthetist.

A rather high mortality rate is associated with this operation, mainly due to pulmonary complications. This is due partly to the fact that coughing is painful with a split sternum and partly because it is ineffectual from the muscular weakness. If, on pre-operative examination, there are any signs of a respiratory infection or a recent history of such, the anæsthetist would be well advised to suggest that thymectomy should be postponed.

My practice is to give prostigmine and atropine only for pre-medication and to induce anæsthesia with intravenous thiopentone. Narcosis is mantained with cyclopropane given with a mask and airway. Intubation is not only unnecessary but tends to cause post-operative tracheitis—a most dangerous complication in such patients. Curare should, of course, never be used.

Post-operative treatment is most important. Although the pleura is seldom opened at operation, a pneumothorax not infrequently develops, and aspiration may be necessary. The initial lesion in pulmonary complications is nearly always a plugged bronchus and as soon as this is detected, the patient should be turned on his sound side, pressure applied over the approximate area of the lesion, and he should be encouraged to cough. It is surprising how often this simple manœuvre will clear a mucous plug. If it fails the intravenous injection of thiopentone to the point of

drowsiness followed immediately by nikethamide, nearly always induces a bout of forceful coughing. Bronchoscopy may occasionally be necessary. Even if no definite physical signs can be detected in the chest, injections of prostigmine and intermittent inhalations of carbon dioxide and air are desirable. In my opinion, the first stage of practically all respiratory complications is mechanical and should be treated as such. If neglected, infection will probably occur in the collapsed lung distal to the plug and treatment becomes much less effective even with modern chemo-therapy.

Cardiac Operations

During operations upon the heart, arrhythmias are frequently produced by the surgical manipulations, in particular, handling of the pericardium and twisting the heart on its axis. The danger is that such arrhythmias may progress to ventricular fibrillation, a condition which is not always amenable even to cardiac massage. It has recently been claimed that the slow intravenous infusion of diluted procaine in doses of from 30 to 70 mg. often restores a normal rhythm, presumably by diminishing the irritability of the cardiac conducting system and causing a shifted pace-maker to revert to the sinus node.

In order to be certain that the anæsthesia does not contribute to any arrhythmia or tachycardia, I have abandoned cyclopropane in such cases in favour of endotracheal nitrous oxide, oxygen and ether. The percentage of oxygen in the mixture should be slightly higher than usual to prevent any possibility of anoxia.

Apart from the removal of foreign bodies in war surgery, the commonest cardiac operations now performed are possibly ligature of a patent ductus arteriosus. Uninfected cases call for little comment but if bacterial endocarditis already exists, the risk is definitely greater. An intravenous drip should be set up in case of severe hæmorrhage but the initial rate of drip should be extremely slow, as these patients often have an increased blood volume. It is interesting to note the immediate rise in diastolic pressure when the ductus is occluded.

I have had no personal experience of anæsthetizing patients for the opposite type of operation where an artificial ductus is formed by an anastomosis between the pulmonary and subclavian arteries in order to create a shunt in certain types of congenital heart defects.

6. TRUFFOT'S STATUE OF JUPILLE, THE SHEPHERD BOY.

AMONG THE POSSESSIONS of Sir John Bland-Sutton specifically bequeathed to the College was a copy of Truffot's fine statue in front of the Pasteur Institute in Paris, which represents the shepherd boy Jupille struggling with a mad dog. This boy, aged 14, in his determination to protect some other children from a mad dog, bravely struggled with it and, with the help of his little brother, tied up the dog's mouth with his whip. He then stunned the dog with his sabot, dragged it to a nearby stream and held it under the water until he was sure it was dead. In the course of the struggle both of Jupille's hands were badly bitten.

The dog's carcass was examined by veterinary surgeons and they decided that the dog was rabid. Jupille was inoculated by Pasteur—the second patient to be so protected—and was saved from Hydrophobia.

Pasteur was so pleased with the brave boy that he found a place for him in the service of the Institute. When Bland-Sutton visited the Pasteur Institute in 1902 he bought an exquisite copy of the statue from Jupille, who was then concierge. He found it in Jupille's rooms and it now adorns the Main Hall of the Royal College of Surgeons.

A.W-J.

SAYINGS OF THE GREAT

" Now that the prestige of science is so high the statement that a great part of medicine still retains the status of an art is often made with a note of apology. Nothing could be less justified by a realistic sense of cultural values."—*Trotter*.

" Sir, the man who has the impatience to interrupt another's remarks has seldom the patience carefully to choose his own."—*Johnson*.

" On earth peace, good will toward men."—*Luke II*, 14.

" Pax hominibus bonæ voluntatis." " Peace to men of good will."—*The Vulgate*.

" I had rather men should ask why my statue is not set up, than why it is."—*Cato*.

" Choose rather to punish your appetites than to be punished by them." —*Epictetus*.

'TRUFFOT'S STATUE OF JUPILLE, THE SHEPHERD BOY.

DIARY FOR JUNE

Tues.	15	5.00	SIR THOMAS FAIRBANK—Abnormalities of the Skeleton.
Wed.	16	5.00	SIR LANCELOT BARRINGTON-WARD—Acute Abdominal Emergencies.
Thurs.	17		First Membership Examination begins.
		5.00	Mr. G. H. MACNAB—Surgery of the Newborn.
Fri.	18		D.P.M. (Part II) and Final L.D.S. (Part II) Examinations begin.
		5.00	MR. H. P. WINSBURY WHITE—Surgery of the Lower Urinary Tract.
Mon.	21	5.00	MR. DENIS BROWNE—Hernia and Undescended Testicle.
Tues.	22	5.00	MR. T. H. SELLORS—Chest Surgery.
Thur.	24	5.00	PROF. A. SORSBY—The Dystrophies of the Retina and Choroid—Recessive Affections.*
Fri.	25		D.P.H. Examination (Part I) and L.D.S. Examination (Special Anatomy and Physiology) begin.
		5.00	PROF. A. SORSBY—The Dystrophies of the Retina and Choroid—Dominant and Sex-linked Affections.*
Tues.	29		Final Membership Examination begins.
		5.00	PROF. H. J. SEDDON—Bone Growth.
		6.15	DR. S. ROWBOTHAM—The Relation of Endocrine Imbalance to Anæsthesia.
Wed.	30	5.00	MR. H. OSMOND-CLARKE—Strains and Sprains.
		6.15	DR. A. H. GALLEY—Continuous Caudal Analgesia in Obstetrics.

DIARY FOR JULY

Thur.	1		D.P.H. (Part II) and Final Membership Examinations begin.
		11.00	Election to Council.
		5.00	MR. J. G. BONNIN—Fractures of the Pelvis.
		6.15	DR. R. P. HARBORD—Anæsthesia in Relation to Shock.
Fri.	2	5.00	MR. B. MCFARLAND—Birth Fractures.
		6.15	DR. G. EDWARDS—Basal Narcosis.
Mon.	5		Oral Examination in Surgery begins.
		5.00	MR. R. WEEDEN BUTLER—Acute Hæmatogenous Osteomyelitis.
		5.00	PROF. H. A. HARRIS—Bone Growth.
		6.15	MR. A. D. MARSTON—History of Anæsthesia.
		6.15	PROF. JOHN BEATTIE—Physiology of Nutrition (1).
Tues.	6	5.00	MR. J. CHARNLEY—Fractures of the Forearm Bones.
		5.00	PROF. J. H. DIBLE—Inflammation and Repair (1).
		6.15	DR. JOHN GILLIES—Anæsthetics for Operations on Cardiovascular conditions.
		6.15	PROF. JOHN BEATTIE—Physiology of Nutrition (2).
Wed.	7	5.00	MR. G. R. GIRDLESTONE—Pott's Disease and Pott's Paraplegia.
		5.00	PROF. J. H. DIBLE—Inflammation and Repair (2).
		6.15	DR. A. C. FORRESTER—Anæsthesia for the Out-patient.
		6.15	DR. C. C. N. VASS—Saliva.
		7.00	Monthly Dinner for Fellows, Members and Licentiates.
Thur.	8	2.15	Election of President, Vice-Presidents and Lecturers.
		5.00	DR. R. B. CATTELL—Carcinoma of the Pancreas*
		5.00	DR. W. A. M. SMART—Hormones.
		6.15	PROF. JOHN KIRK—Anterior Triangles.
Fri.	9		D.O.M.S. Examination (Part 1) begins.
		5.00	MR. K. I. NISSEN—Non-tuberculous Affections of the Hip Joint in Childhood and Adolescence.
		5.00	PROF. E. J. KING—Blood Chemistry.
		6.15	DR. G. ORGANE—The Assessment of Anæsthetic Risk.
		6.15	PROF. W. T. ASTBURY—Collagen and Keralin.

* Not part of courses.

Mon.	12	5.00	MR. CAPENER—Orthopædic Appliances.
		5.00	MR. R. J. LAST—Anatomy and Development of the Maxillary Antrum.
		6.15	DR. H. G. EPSTEIN—Some Aspects of Physics in Anæsthesia.
		6.15	PROF. D. T. HARRIS—Pain.
Tues.	13	5.00	MR. R. FURLONG—Injuries of the Wrist and Carpus.
		5.00	DR. D. LONG—Penicillin in Acute Oral and Naso-Pharyngeal Infection.
		6.15	DR. R. JARMAN—The Chemistry of Common Anæsthetics.
		6.15	DR. K. C. RICHARDSON—General Histology in Relation to the Oral Cavity.
Wed.	14		Primary F.R.C.S. Examination begins.
		5.00	MR. A. N. BIRKETT—Fractures of the Cervical Spine.
		5.00	PROF. JOHN KIRK—Fascial Planes.
		6.15	DR. B. L. S. MURTAGH—Avoidable Accidents in Anæsthesia.
		6.15	DR. R. L. WATERFIELD—Blood—Physiological Aspect (1).
Thur.	15	5.00	PROF. T. P. MCMURRAY—Derangements of the Knee Joint.
		5.00	DR. J. SHORT—Development of the Jaws.
		6.15	DR. R. WOOLMER—Diagnostic and Therapeutic Uses of Anæsthetics.
		6.15	DR. R. L. WATERFIELD—Blood—Physiological Aspect (2).
Fri.	16		D.O.M.S. Examination (Part II) begins.
		5.00	PROF. A. F. JACKSON—Charles Tomes Lecture—Growth and Development from the Clinical Aspect of Orthodontics.*
Mon.	19	5.00	PROF. S. L. BAKER—General Pathology of Bone (1).
		6.15	DR. C. H. TONGE—Surgical Anatomy in Relation to Regional Anæsthesia.
Tues.	20	5.00	PROF. S. L. BAKER—General Pathology of Bone (2).
		6.15	DR. C. H. TONGE—Anatomy of the Oral Cavity.
Wed.	21	5.00	MR. E. B. MANLEY—The Histology of the Dental Tissues (1).
Thur.	22		D.T.M. & H. Examination begins.
		5.00	PROF. R. J. BROCKLEHURST—Taste.
		5.00	PROF. JOHN BEATTIE—The Changes in Volume and Distribution of Body Water under conditions of Stress.*
		6.15	MR. E. B. MANLEY—The Histology of the Dental Tissues (2).
Fri.	23	5.00	MR. E. B. MANLEY—The Histology of the Dental Tissues (3).
		6.15	MR. R. T. PAYNE—Acute Infection of the Salivary Glands.
Mon.	26	5.00	PROF. H. A. HARRIS—Clinical Anatomy of Thorax.
		6.15	MR. A. BULLEID—Bacteriology of the Mouth.
Tues.	27	5.00	PROF. D. T. HARRIS—The Autonomic Nervous System.
		6.15	DR. A. C. COUNSELL—Ætiology of Cysts of the Mouth.
Wed.	28	5.00	DR. M. W. CARR—Charles Tomes Lecture—Acute Infections of the Face and Neck of Dental Origin.*
Thur.	29	5.00	PROF. SHAFIK SHELABY—Hunterian Lecture—Amœbic Liver Abscess.*
		5.00	DR. W. A. M. SMART—Circulation.
		6.15	PROF. E. P. WRIGHT—Repair of Connective Tissues.
Fri.	30	5.00	DR. C. REID—Respiration and Anoxœmia.

* Not part of courses.

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